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HISTOLOGY OF FISTULAS FOLLOWING ANTERIOR SCLERECTOMY *

S. HOLTH, M.D.

OSLO, NORWAY

In my paper on iridencleisis as an operation in glaucoma, published in the December, 1930, issue of the ARCHIVES,[1] I said that I had abandoned anterior sclerectomy in spite of its giving "an immediate sometime brutal fall of tension." I gave it up not only on account of the relatively frequent late infections, but also because in many cases after several years the defect in the sclera may be completely obliterated by solid and impermeable scar tissue. The tension is then often raised again, and not lowered by miotics.

After anterior sclerectomy and after iridencleisis in rabbits the scar tissue never causes a permanent subconjunctival fistula and accordingly cannot be used in this study; nor can the scars be studied that follow the same operation in human beings with glaucoma absolutum dolorosum, in whom the eye must be enucleated later on account of recurring pain.

The histologic process in the scar of a subconjunctival fistula can be studied only after the death of the patient, a long time after successful operation for primary glaucoma. In nine such cases I showed the subconjunctival fistula of iris tissue in serial sections of the scars at the death of the patient, from eight months to six years after successful iridencleisis.[2] Five of these cases were illustrated in the December, 1930, issue of the ARCHIVES.

I shall now show the histologic process of the fistula following a subconjunctival anterior sclerectomy in three cases in which limbal sclerec-

* Submitted for publication, March 28, 1931.

* Read at the discussion on the operative treatment for glaucoma at the Eighth Scandinavian Ophthalmological Congress, Helsingfors, Finland, June 25 to 27, 1931.

1. Holth, S.: Iridencleisis cum Iridotomia Meridionali: An Operative Method Both in Acute and in Chronic Primary Glaucoma, Arch. Ophth. **4**:803 (Dec.) 1930.

2. Holth: Anatomische Untersuchung der Operationsnarben und der aplanierten Papillenexkavation nach erfolgreichen Glaukomoperationen (Iridencleisis, Sclerectomia limbalis nach Holth oder Elliot, Sclerecto-Iridencleisis), Ber. d. Ophth. Gesellsch., Heidelberg, 1913, p. 355; Anatomical Examination of Six New Cases of Subconjunctival Fistula Scars from Five Months to Six Years After Successful Iridencleisis or Limbal Sclerectomies in Chronic Glaucoma, Brit. J. Ophth. **6**:10, 1922.

tomy had been carried out with the best possible result about three years before death. In two of these cases the operation consisted of my limbal sclerectomy with punch forceps (1908). In the third case Elliot's trephining was carried out. All these operations were combined with iridectomy without any trace of iris inclusion. Five illustrations will be given, together with critical comment.

REPORT OF CASES

CASE 1.—Mrs. M. G., aged 79, underwent my limbal punch forceps sclerectomy combined with an ordinary iridectomy on the left eye on March 16, 1909. In the serial section (fig. 1) made after her death on April 30, 1912, one can see the scleral defect covered with pigment epithelium grown out from the root of the iris and from the processus ciliares. Before operation the intra-ocular tension was 43 mm. of mercury, in spite of miotics; after operation, on an average, 15 mm.

Figure 2 shows that the microtome has torn the pigment epithelium away from the posterior wall of the scleral defect and deposited it in front of the anterior

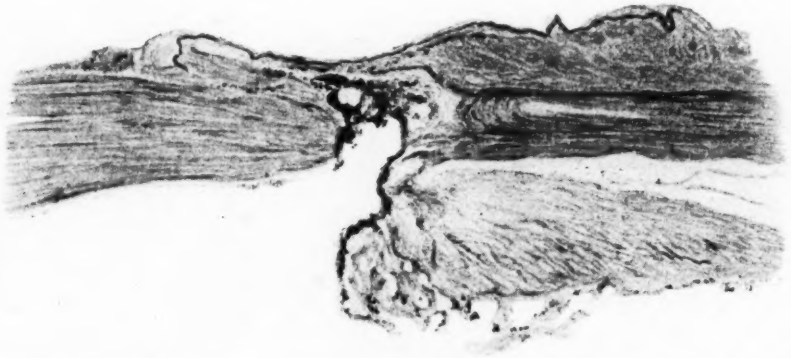

Fig. 1 (case 1).—Axial vertical section through the sclerectomy defect in left eye (somewhat laterally to the center) following Holth's limbal punch forceps sclerectomy combined with an ordinary iridectomy.

wall of the ciliary body. The dark and dense subconjunctival scar tissue is perforated in the posterior part by a light space composed of a fine lax connective tissue poor in cells.

Figures 1 and 2 show a point of great importance. When the walls of the scleral defect are covered by immigrated uveal pigment epithelium, scleral scar formation is hindered, as is any considerable immigration of subconjunctival scar tissues into the scleral defect. Therefore twice during the year 1912 I tried to combine anterior sclerectomy with voluntary iridencleisis, a fact of which L. Weekers [3] recently reminded me. Among my preparations demonstrated in Heidelberg in 1913 was one from the scar following sclerecto-iridencleisis performed on May 8, 1912, under a trephining, six months before the

3. Weekers, L.: L'action hypotonisante, chirurgicale et médicamenteuse, dans la thérapeutique antiglaucomateuse, Acta ophth. **8**:253, 1930.

death of the patient.[4] I soon abandoned this technic, however, as did Gunnufsen [5] who tried the combined procedure with trephining in four cases, using a difficult technic. The spontaneous immigration of the uveal pigment epithelium after the iridectomy is more reliable than the inclusion of the whole thickness of the iris under the thin conjunctiva in the limbus itself. This immigration is, however, not certain to take place, although it often does.

CASE 2.—In Miss E. N., who died on May 6, 1915, at the age of 57½, a limbal sclerectomy had been done upward on the right eye with Holth's punch forceps, 3 mm. wide, on Jan. 17, 1912. The incision measured 1.5 by 3 mm. A basal iridectomy

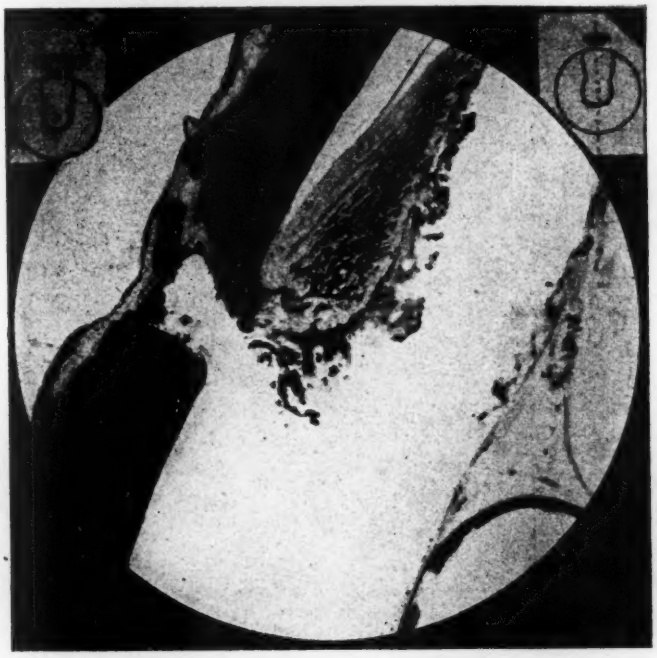

Fig. 2 (case 1).—Axial vertical section passing exactly through the center of the scleral defect in left eye; × 18. In upper left-hand corner is a diagram of Holth's limbal punch forceps sclerectomy (1908); in upper right-hand corner, a diagram showing the somewhat diminished size of the scleral defect three years after operation.

was also done. The sclerectomy defect was first seen through a thin glassy bubble as a black opening; after a time it became somewhat smaller and was divided in the middle into two halves by a nearly meridional and slightly oblique gray cicatricial bridge in the depth of the opening. Figure 3 shows the anterior (corneal) wall of the defect lined with a thin layer of connective tissue the fibers of which run in a vertical direction to the course of the sclerocorneal fibers. This

4. Holth (footnote 2, first reference).

5. Gunnufsen, T.: Ueber die operative Behandlung des primären Glaukoms, Klin. Monatsbl. f. Augenh. **57**:283, 1916.

thin layer of connective cicatricial tissue is lined with endothelium from Descemet's membrane, but the endothelium does not continue under the internal surface of the conjunctival covering or on the posterior (ciliary) wall where the scleral fibers end free without any sign of a reactive process. The epithelium of the covering conjunctiva is normal; the conjunctival connective tissue is somewhat denser and richer in cells than elsewhere away from the operative field. The deep gray cicatricial bridge develops from this subconjunctival scar tissue. The light area near the cornea shows lax connective tissue poor in cells. Before operation the intra-ocular tension was 61 mm. of mercury, in spite of miotics; on Jan. 30, 1915, the tension was 18 mm. without miotics.

Fig. 3 (case 2).—Axial vertical section through the scar after a limbal sclerectomy and a basal iridectomy on the right eye; × 18.

In figure 3, I especially want to call attention to the fact that the scleral fibers of the posterior (ciliary) wall, made with a small stop keratome, are completely free from reaction. The same fact was noted by Thompson-Henderson [6] in the iridial tissues at the edge of an iridectomy. "For even years after, the cut surface appears as if the operation had been performed post mortem."

It was this discovery of Thompson-Henderson which in 1908 made me hope for somewhat similar results from sclerectomy. In this opera-

6. Thompson-Henderson: The Histology of Iridectomy, Ophthalmic Rev. **26**: 191, 1907.

tion, however, a slow reactive process is the rule. It is, however, possible that the complete lack of a reaction in the posterior wall of the scleral tissues shown in figure 3 and in the endothelial covering on the anterior wall would have prevented the deep gray scar bridge from filling out the whole scleral defect even if the patient had lived much longer than three years after the sclerecto-iridectomy.

CASE 3.—E. N. (the same patient as in case 2) underwent a limbal sclerectomy upward on the left eye with a 2 mm. trephine, on Jan. 9, 1912; a basal iridectomy was done. During the first year the scleral defect was seen through a thin glassy

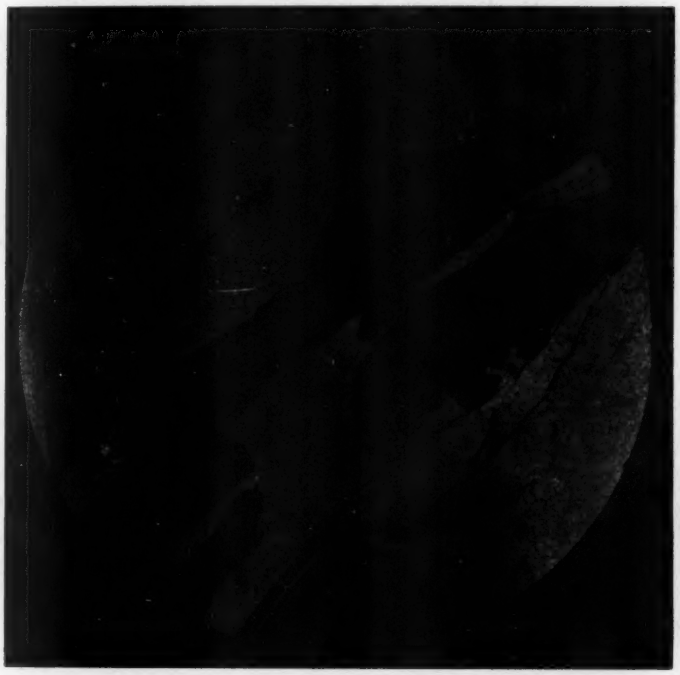

Fig. 4 (case 3).—Axial vertical section through the center of the scar in the left eye after a limbal sclerectomy upward with a 2 mm. trephine, combined with a basal iridectomy; × 18.

conjunctival bubble as a black round hole 2 mm. in diameter. After a time the hole became smaller, and on Jan. 30, 1915, it appeared as a black subconjunctival opening 0.75 mm. wide in the middle of a gray field corresponding to the original 2 mm. trephine hole. Figure 4 shows the conjunctiva with normal epithelium passing over the defect. The anterior (corneal) wall of the original defect is lined with a thin layer of dense connective tissue, somewhat rich in cells, with vertically running fibers and lined with endothelium from Descemet's membrane; this endothelium is not seen elsewhere on the walls of the defect. The deep subconjunctival layers consist of a denser connective tissue, somewhat rich in cells, with fibers running chiefly in a meridional direction; near both edges of the light central region the fibers in the deeply stained area run in a circular manner. In the anterior (corneal) wall of the scleral defect the denser, darkly stained cica-

tricial tissue originates exclusively in the subconjunctival area; in the posterior (ciliary) wall of the defect, the darkly stained cicatricial tissue consists of two layers, the uppermost one proceeding from the subconjunctival tissue and the deeper one from the scleral wall itself; on the border between the two layers episcleral vessels are seen. In the middle of the darkly staining tissues which I have called "the subconjunctival cicatricial covering" is seen a light area of lax connective tissue with extremely fine, large-meshed fibrils and a few scattered cells. This tissue is continued upward toward the surface, even under the normal epithelium. Before operation the intra-ocular tension was 51 mm. of mercury, in spite of miotics; the first year after trephining, about 15 mm., and on Jan. 30,

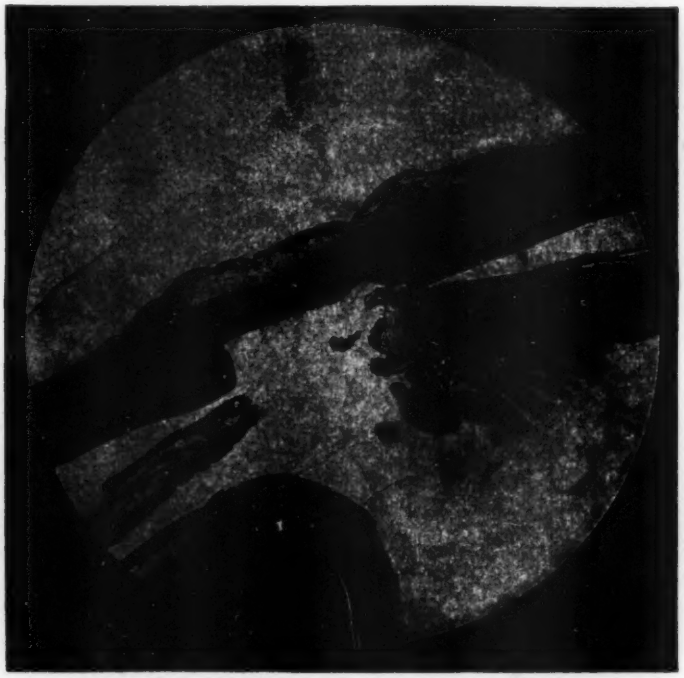

Fig. 5 (case 3).—Vertical sagittal section taken 0.5 mm. temporally to the section shown in figure 4.

1915, 29.5 mm. Pilocarpine, 2 per cent, was then prescribed for instillation at bed time.

In figure 5 the dense darkly stained subconjunctival cicatricial tissue covers the scleral defect completely.

If the patient in case 2 had lived longer than three and one-third years after the trephining I am sure that the central black hole, 0.75 mm. in size, would have been transformed into a gray scar, and that all the serial sections would have been as in figure 5. Clinically I have repeatedly noted this transformation after trephining. I have also seen that the increased tension under these circumstances is insufficiently influenced by miotics.

CONCLUSIONS

1. The best conditions under which the subconjunctival fistula following anterior sclerectomy may remain open for many years are provided when the walls of the defect are covered by uveal pigment epithelium.

2. To gain this object it is necessary that an ordinary or a basal iridectomy be performed immediately after the sclerectomy. The immigration of the pigment epithelium cannot be depended on, however, and it never takes place after an isolated anterior sclerectomy. Exceptions to this rule are cases in which there is a postoperative prolapse of the pupillary edge.

3. The practice of combining anterior sclerectomy with iridencleisis must, however, be discouraged. The spontaneous immigration of the isolated pigment epithelium, which is often seen, is less risky than inclusion of the whole thickness of the iris under the thinnest conjunctiva at the limbus corneae.

4. After my iridencleisis through an extralimbal, subconjunctival keratome incision the iridial fistula will regularly open from 2 to 3 mm. from the limbus under a thicker cover of conjunctiva.

5. The scleral beak (fig. 3) protects the ciliary body against seconddary atrophy.

6. Iridencleisis cum iridotomia meridionali should always be performed alone—never combined with anterior sclerectomy.

THE METABOLISM OF THE EYE

II. CLINICAL APPLICATIONS*

W. S. DUKE-ELDER, M.D.
LONDON, ENGLAND

In a preceding lecture, I endeavored to show that the metabolism of the eye could, with some degree of justification, be considered under the heads of four equilibria. In this lecture, I shall endeavor to show with what profit this conception may be applied. The physiologic conception of the metabolism of the eye was crudely, and also of necessity, briefly expressed. In one sense it may have seemed somewhat complicated, and in another it may have seemed to carry with it an air of simplicity. It is to be remembered, however, that it is by no means final. It represents merely the beginning of a new point of view, an attempt to look at things in as fundamental a way as possible, to coordinate a complexity of observations by simplifying them in a general theory. It is to be remembered (in the words of C. D. Broad) that there are two kinds of simplicity: a lower type whereby one isolates a small fragment of one's problem and ignores all the other fragments that do not fit in with it, and a higher simplicity which deals with the general plan on which the whole is constructed; but in order to pass from the lower to the higher type, one must traverse a region of great complexity, wherein one must confront the lower simplicity with all the awkward facts it has ignored. So far as the present subject is concerned, it is still in this somewhat difficult and unsatisfactory stage.

The first clinical problem with which I shall deal is that of intra-ocular pressure and glaucoma. By the theory of equilibria, changes in the pressure of the eye should be reduced to simple terms. These changes may be conveniently divided into three categories: First, external pressure on the globe, which, in the usual conditions of life, resolves itself into the action of the extra-ocular muscles. This question has already been considered from the physiologic standpoint, but there are three points that probably merit further attention from the clinical aspect.

In investigating changes of intra-ocular pressure, in addition to employing the classic methods of animal experimentation, I have been accustomed to make use of a preparation whereby the eye is artificially perfused with the apparatus seen in figure 1. A dog is bled, and the whipped blood is gathered into reservoirs (K^1, K^2, K^3) which are maintained at a constant temperature by an electrically con-

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* The Lucien Howe Memorial Lectures (1930) delivered at the University of Harvard.

trolled thermostat (*r*, *w*). This can be pumped up through an artificial heart pump (*j*), the amplitude of whose beat is varied at will by an adjusting mechanism (*M*, *N*, *O*), while the temperature of the blood is still maintained by passing it through coiled tubing (*G*, *H*) in the enclosing water-bath, heated (*K*, *L*) similarly to the reservoirs. After the blood leaves the pump, it passes an apparatus (*P*, *Q*, *R*, *S*, *T*) which maintains its pressure level constant at any desired height by short-circuiting any excess through *Q* back to the reservoirs should it rise above that height. Passing this, the pressure is recorded by the manometer (*D*).

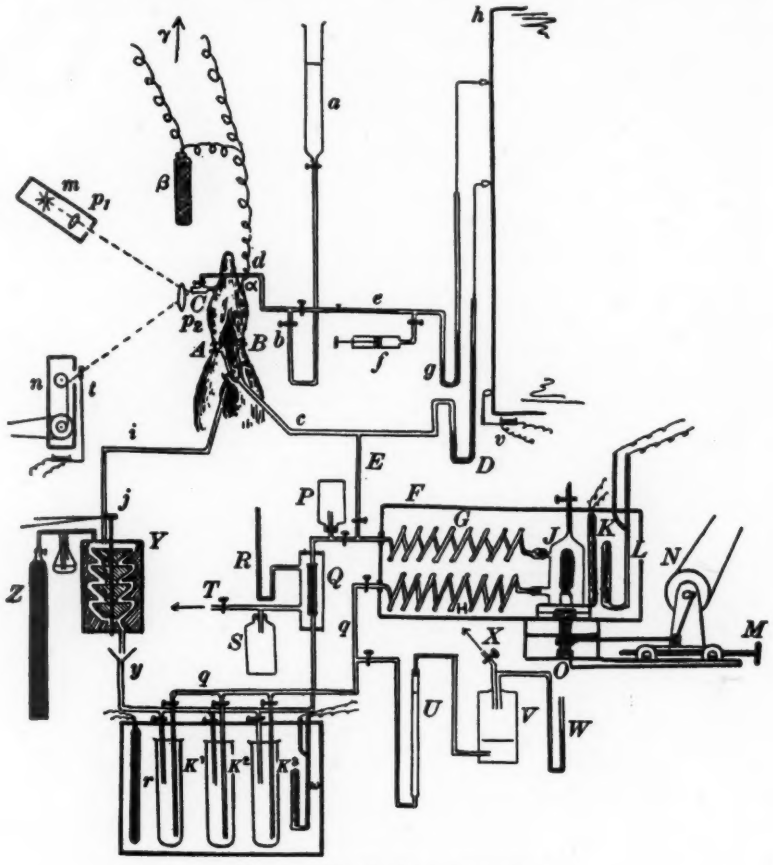

Fig. 1.—Perfusion apparatus.

The left carotid artery of a second dog is connected with this circuit (*B*); at this stage the head is perfused through the right carotid by the dog's own blood, and through the left by the artificial circulation. The right carotid is then similarly connected (at *A*), and the head is now perfused artificially without having allowed a cessation of the circulation at any time. The blood leaving the head is gathered from the superior vena cava, and, passing through an artificial lung (*Y*, *Z*), where it is aerated, is returned to the reservoirs whence the circuit is continued. To maintain arterial tone, a constant and minute supply of epinephrine is added to the circulating blood (*U*, *V*, *W*). The intra-ocular pressure is recorded from the right eye manometrically, and, as an aid in analyzing vascular reactions, the temperature in the anterior chamber of the left eye is recorded by a

thermopile needle and a galvanometer (α, β, γ). In this way the head is artificially perfused without any cessation in the circulation in such a manner that all extraneous factors are kept absolutely constant and pressure changes occurring in the eye alone can be studied uncomplicated by systemic effects elsewhere; by analyzing results obtained in this way and comparing them with results obtained in intact animals, a coordinated picture can be drawn. There is no question of the continued vitality of the head; an anesthetic has to be given continuously (chloralose was employed), a corneal reflex can be readily elicited, pupillary reactions are maintained, and the circulation can be observed ophthalmoscopically or followed in the pulse beat of the record of the intra-ocular pressure.

The intra-ocular pressure was recorded by the manometer illustrated in figure 2. A cannula (D) is inserted into the eye, and the absolute height of the pressure of the eye recorded by a mercury manometer (B), a compensatory apparatus,

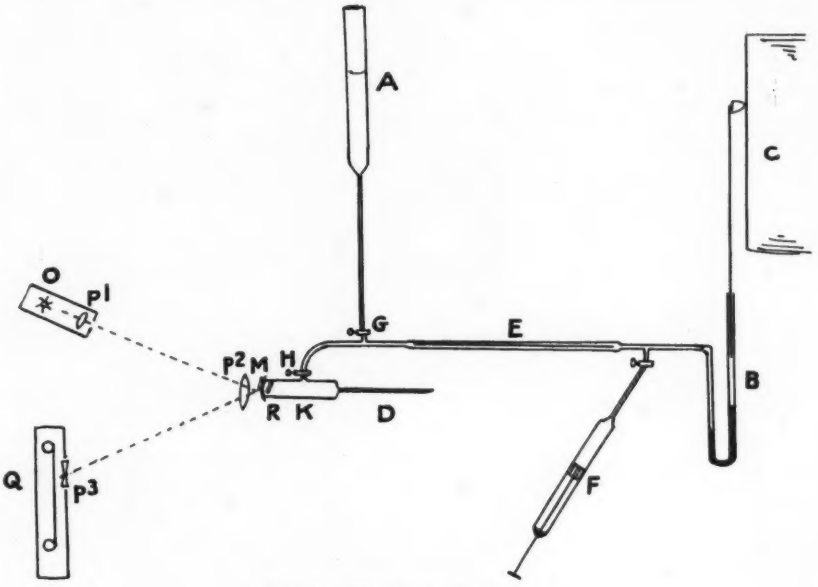

Fig. 2.—Ocular manometer.

whereby an air bubble in the capillary tube (E) was equilibrated by an accurately controlled pressure syringe (F), being interposed so that the volume of intra-ocular fluid is kept constant in all circumstances. Once equilibrium is established the stopcock (H) is closed, and an optical method is employed to follow the subsequent variations of pressure. A glass chamber (K) is closed distally by a rubber membrane (M) to which a minute mirror is attached. This reflects a beam of light from a source (O) through an optical system (P^1, P^2, P^3) to a moving photo-film in the camera (Q); while a second mirror (R) fixed to the glass chamber records a second tracing which acts as a base line. Events in the eye are correlated with events in the general circulation by two synchronized time markers (N, M).

A matter of considerable clinical importance with regard to the extra-ocular muscles is the effect of their tone on the normal pressure of the eye. This is seen in the fall of intra-ocular pressure which occurs

on the administration of curare, which paralyzes these muscles. When this is done in an intact animal, it is difficult to dissociate the fall in ocular pressure from the accompanying fall in general blood pressure. But when the blood pressure in the perfused animal is kept constant (fig. 3), the lack of muscular tone reduces the pressure in the eye to about half its original level. The fact that the normal intra-ocular pressure depends to such an extent on muscular tone is a point that probably has not received sufficient attention before. There is another point in

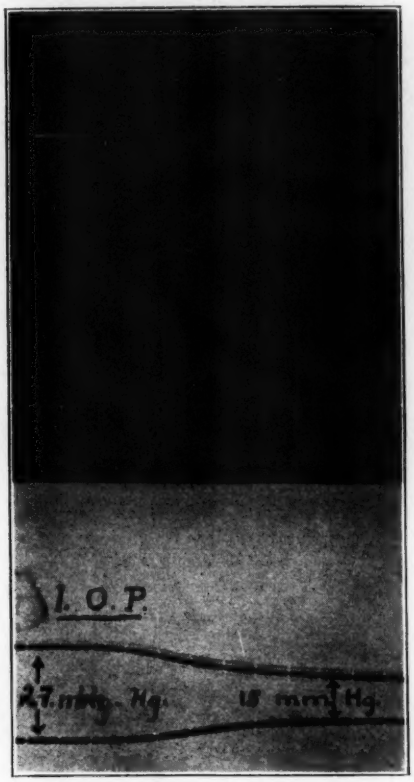

Fig. 3.—The effect of curare (2 cc. of 2 per cent) on the perfused dog's head. The upper curve is the blood pressure and the lower the intra-ocular pressure.

connection with the extra-ocular muscles which is of clinical importance, and which will be evident from figure 11 in part I of this paper. It demonstrates the effect on the pressure of the eye of a contraction of the orbicularis muscle caused by stimulation of the facial nerve, and it provides an apt demonstration of the danger of attempting an intra-ocular operation without adequate regional akinesia. The influence of the recti muscle is as important. Figure 10 in part I shows the effect on the pressure of the eye of a contraction of these muscles as induced

by choline. It demonstrates the danger of the performance of intra-ocular operations, such as extraction of cataract, without suitable anesthetization of these muscles as well as the facial muscles, for in obeying the instructions to look down (and so on) the patient must raise the pressure of the eye to the extent seen in figures. The only adequate technic, from the physiologic point of view, is to paralyze both sets of muscles—orbicularis and recti—and to employ a stay stitch to control movements of the eyes. Finally, the action of these muscles as a whole, it will be remembered, has an intensely important effect in determining the pressure circulation of the intra-ocular fluid, and it is probably this factor that is responsible for the diurnal variation in pressure that forms so valuable a sign in early glaucoma. It has been established that in this disease the pressure is at its highest in the early hours of the morning before active movements commence and at its lowest at the end of

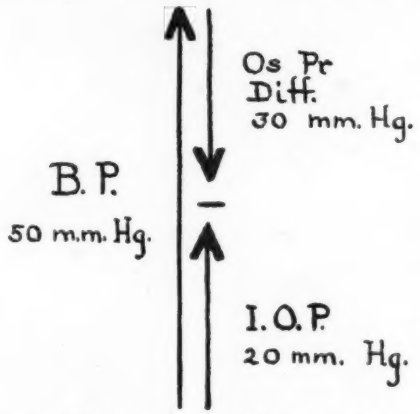

Fig. 4.—The pressure equilibrium of the eye.

the day's activities, the phenomenon being due to the action of the extra-ocular muscles which carry on a massage-like action during the day.

The second set of circumstances by which the intra-ocular pressure can be affected is an alteration of the equilibrium level. It will be remembered from the earlier lecture that the pressure equilibrium of the eye was determined by the equilibrium between the blood pressure in the capillaries and the difference between the osmotic pressure of the plasma and of the intra-ocular fluid (fig. 4). This equilibrium can obviously be changed by an alteration either of blood pressure or of osmotic pressure. I do not intend to go deeply into the question of alterations in the blood pressure, as this subject is already well known. It is to be remembered, however, that so far as clinical circumstances are concerned, a high blood pressure frequently means a low capillary pressure, owing to the accompanying sclerosis in the arterioles—a circumstance that explains the fact that the intra-ocular pressure does not

by any means follow the blood pressure. It might be of interest, however, in passing, to demonstrate some illustrations of the effect of various drugs on the pressure of the eye in this connection.

Epinephrine in small doses dilates the capillaries, increases their permeability and raises the intra-ocular pressure (fig. 5), while in large doses it constricts the small vessels and lowers the intra-ocular pressure (fig. 6). This is complicated by the fact that in any dose it tends to stimulate the plain muscle of the orbit and thus to raise the intra-ocular pressure.

Solution of pituitary constricts the arterioles and lowers the intra-ocular pressure, an action which also is complicated by the fact that it

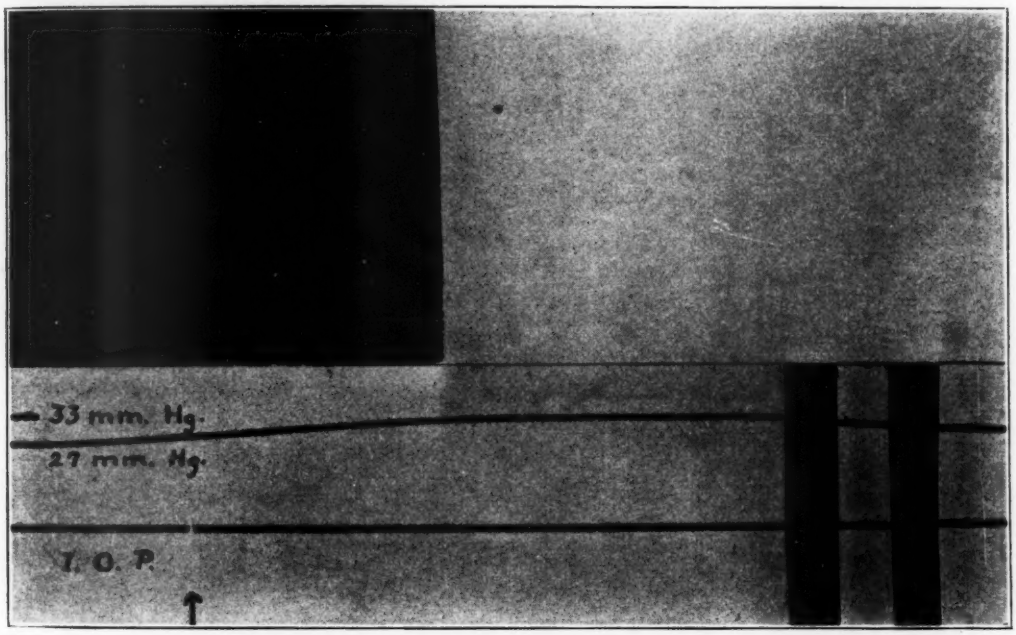

Fig. 5.—The effect of 0.1 cc. of 1 : 10,000 epinephrine on the perfused dog's head.

stimulates the plain muscle of the orbit, thus tending to raise the intra-ocular pressure (fig. 7).

Histamine varies in its action on the small vessels of the eye. If these vessels have a considerable tone, histamine dilates them, increases their permeability and increases the intra-ocular pressure (fig. 8); if, on the other hand, vascular tone is lacking, histamine constricts the arterioles and lowers the intra-ocular pressure (fig. 9). This variation in the effective action of epinephrine and histamine, depending on the active dose and the state of vascular tonicity, accounts for the great inconstancy met with in the treatment for glaucoma by the so-called "glaucosans."

Fig. 6.—The effect of 0.1 cc. of 1 : 1,000 epinephrine on the perfused dog's head. The spacings in the tracing of the intra-ocular pressure represent two minute intervals.

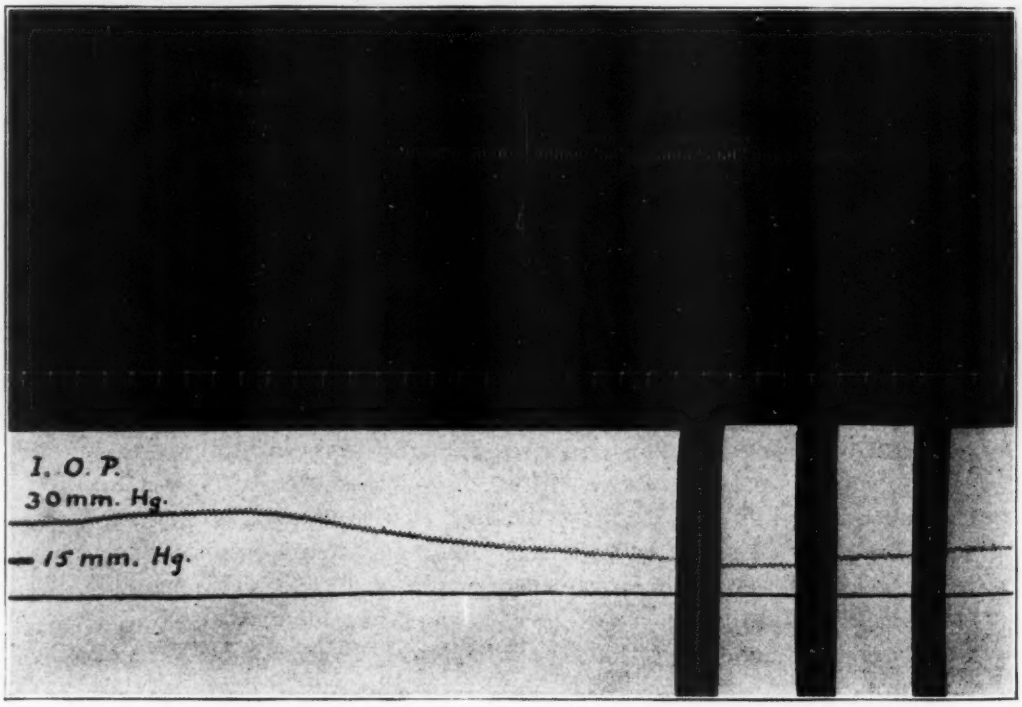

Fig. 7.—The effect of 0.5 cc. (10 international units per cubic centimeter) of solution of pituitary on the perfused dog's head.

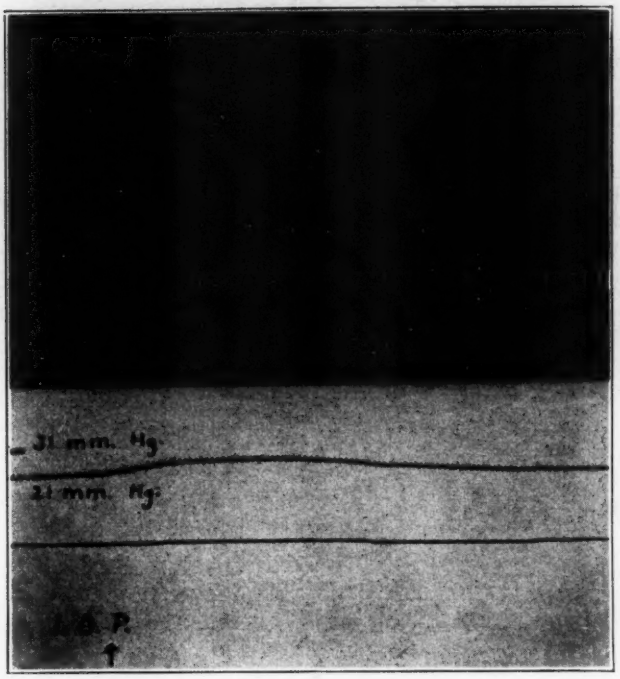

Fig. 8.—The effect of 1 cc. of 1 : 100,000 histamine on the perfused dog's head with constant epinephrine in the perfusing blood.

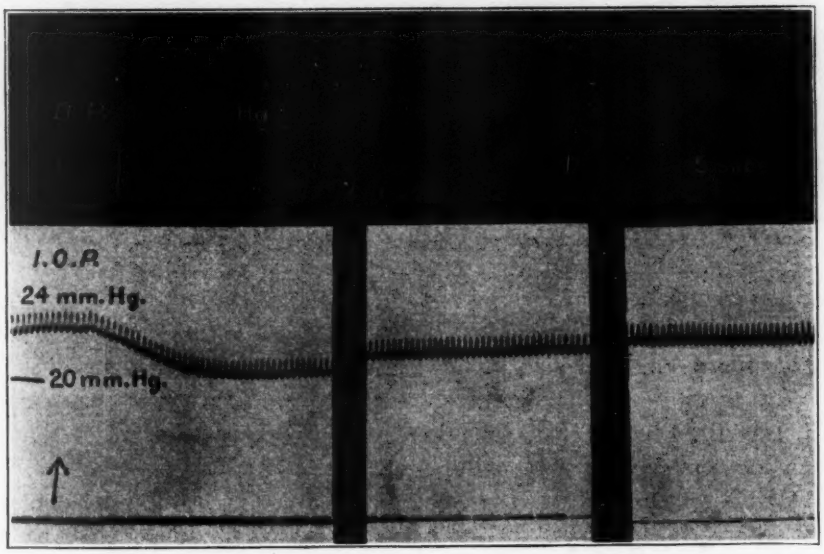

Fig. 9.—The effect of 0.5 cc. of 1 : 10,000 histamine on the perfused dog's head (capillary tone poor owing to lack of constant epinephrine). The spacings in the tracing of the intra-ocular pressure represent two minute intervals.

Atropine dilates the minute vessels of the eye and increases their permeability, thus raising the intra-ocular pressure, an action which is frequently complicated by an accompanying relaxation of the plain muscle of the orbit, which tends to lower the intra-ocular pressure (fig. 10). Physostigmine (eserine) dilates the small vessels and increases their permeability and, in addition, increases the tone of the voluntary muscles of the orbit, both actions tending to raise the pressure in the eye (fig. 11). If, however, the channels of exit of the intra-ocular fluid are obstructed at the angle of the anterior chamber, these two drugs have the opposite effect, physostigmine opening the channels and lowering the intra-ocular pressure, and atropine occluding them and raising the intra-ocular pressure.

The equilibrium level can also be altered by changing the difference in osmotic pressure between the aqueous humor and the blood plasma. Since the capillary walls are freely permeable to crystalloids, it is evident that these substances will rapidly diffuse from the blood to the eye, so that any change which can maintain a permanent difference in osmotic pressure between the two fluids must be confined to the colloid content. The blood contains a considerable amount of colloids, and the aqueous merely traces. It follows that if the colloid content of the blood is increased, the difference between it and the intra-ocular fluid will be increased and the intra-ocular pressure will fall (fig. 12). Conversely, if the colloids in the blood diminish, it will resemble more nearly the aqueous, and the intra-ocular pressure will rise (fig. 13). Since, however, the colloid content of the blood is a relatively constant quantity, it is obvious that such a factor will have little clinical influence. The osmotic pressure of the colloids in the aqueous humor can be changed only by increasing them, and when this is done, since the difference between the aqueous humor and the blood is thereby diminished, the intra-ocular pressure must rise (fig. 14). This is of considerable importance, for an increase in the proteins of the aqueous occurs in many clinical conditions, such as iritis or cyclitis, and this forms a factor in the occurrence of secondary glaucoma. As a cause of the rise of pressure it is, of course, complicated by a tendency toward blocking of the filtration channels at the angle of the anterior chamber by a richly albuminous and viscous fluid, a matter which has been discussed.

The third set of circumstances that may influence the intra-ocular pressure is a change in the volume of the contents of the globe, for, since the contents of the eye are enclosed within a relatively feebly distensible case, any change in volume is rapidly converted into a change in pressure. The change of volume may involve either the coats of the eye or the aqueous humor, the lens and the vitreous body. With regard to the first of these, figure 15 shows that although the sclerotic does swell and shrink, for example, with changes in the reaction of the sur-

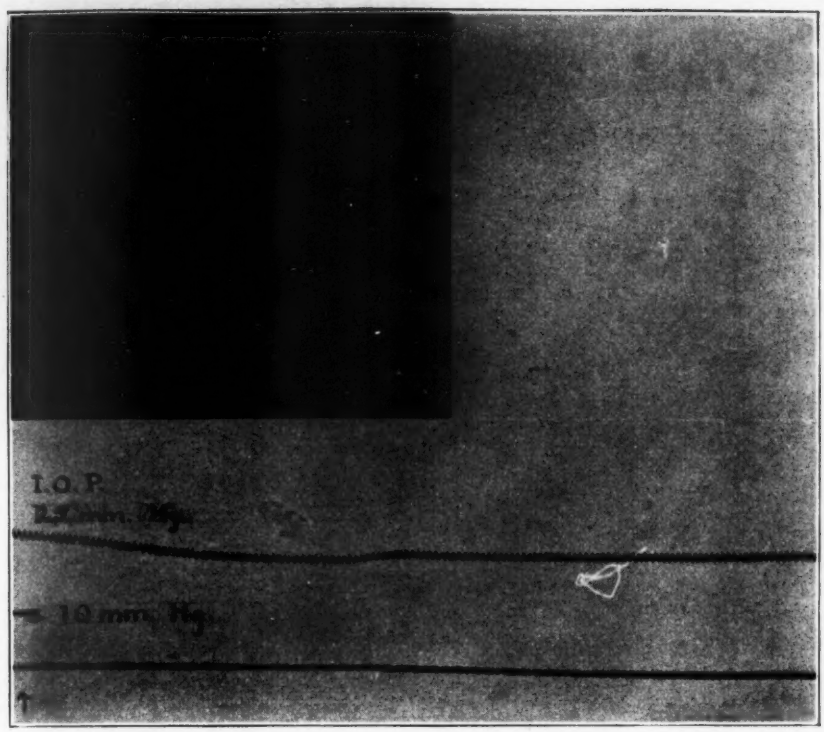

Fig. 10.—The effect of 0.3 cc. of 1 per cent atropine on the perfused dog's head. The fall in pressure is due to the contraction of Müller's muscle. The rise in the middle of the tracing of the intra-ocular pressure represents a capillary dilatation.

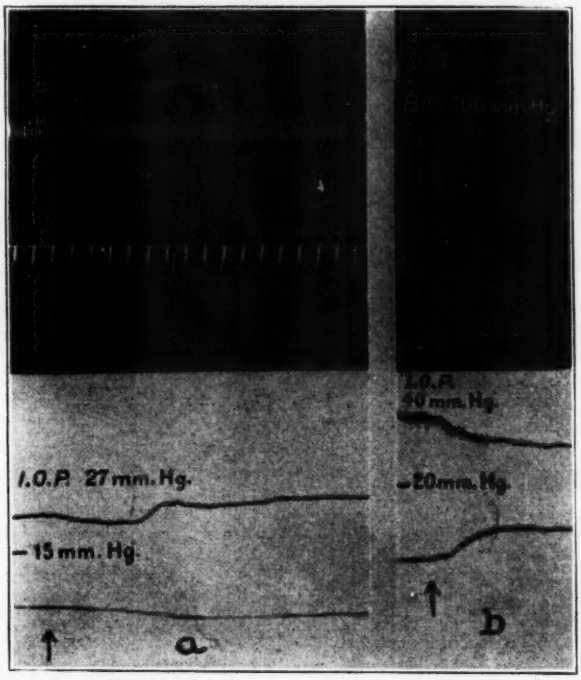

Fig. 11.—(*a*) The effect of 1.5 cc. of 1 per cent physostigmine on the perfused dog's head. The irregular rise is due to increased tonicity of the recti. (*b*) The effect of 2 cc. of 2 per cent curare after 1 cc. of 1 per cent physostigmine on the perfused dog's head. The higher intra-ocular pressure after curare when compared with figure 3 represents the rise in pressure due to capillary dilatation after the muscular effects of physostigmine have been abolished by curare.

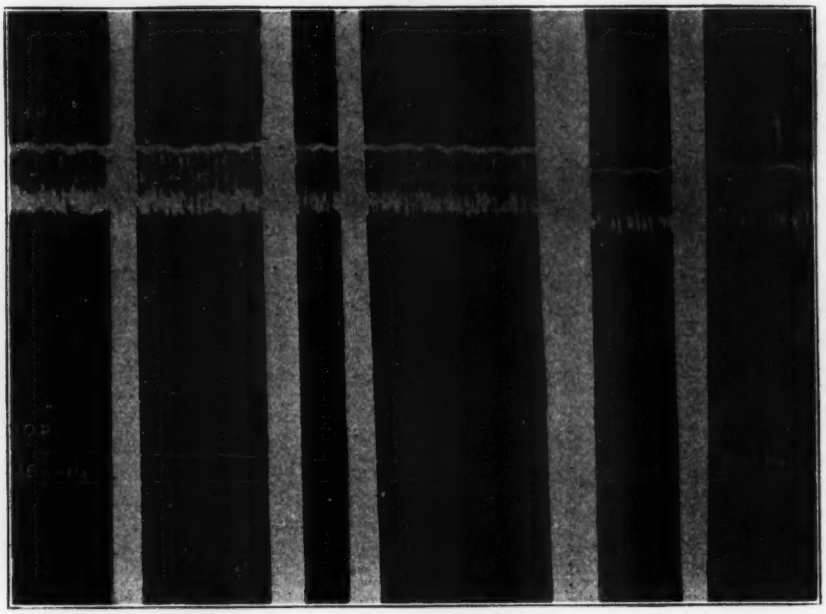

Fig. 12.—The fall of intra-ocular pressure with an increase in concentration of colloids in the blood (perfused eye; mercury manometer record); 15 per cent of gum arabic was added to the perfusing blood.

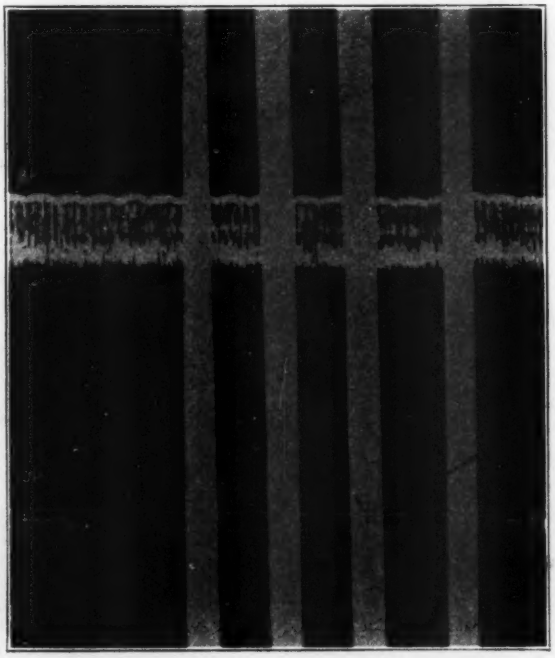

Fig. 13.—Rise of intra-ocular pressure with a diminution in the concentration of colloids by mixing two-thirds blood with one-third physiologic solution of sodium chloride. The condition of the experiment is the same as in figure 12.

Fig. 14.—At *a*, the aqueous humor was replaced by physiologic solution of sodium chloride; no effect on the intra-ocular pressure was produced. At *b*, perfusion with gum arabic solution (15 per cent) resulted in a rise of intra-ocular pressure. During the actual perfusion, the intra-ocular pressure was maintained constant by means of a manometer.

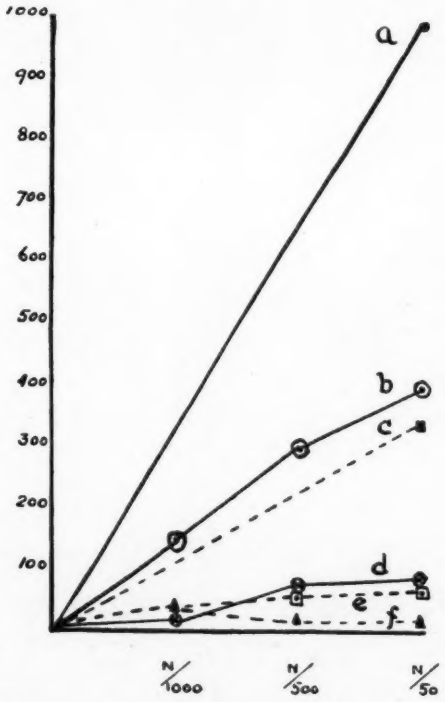

Fig. 15.—Turgescence of the sclerotic in solutions of various activities by weight: (*a*) dialyzed salt-free sclera in acid; (*b*) normal sclera in acid; (*c*) dialyzed salt-free in alkali; (*d*) acid in physiologic solution of sodium chloride; (*e*) normal sclera in alkali; (*f*) alkali in physiologic solution of sodium chloride. The ordinates denote the percentage of turgescence by weight in twenty-four hours; the abscissae, the percentage strength in solution of acid and alkalis.

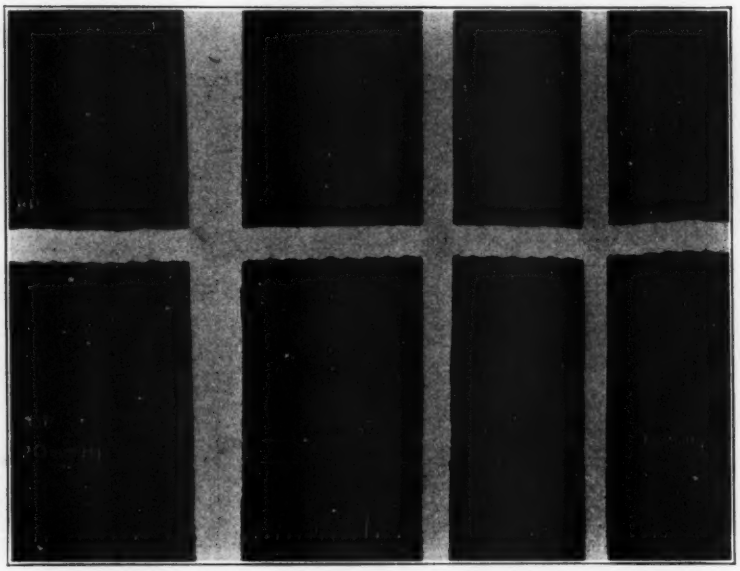

Fig. 16.—Fall of intra-ocular pressure on increasing the concentration of crystalloids by mixing two-thirds blood and one-third 20 per cent saline in perfusion.

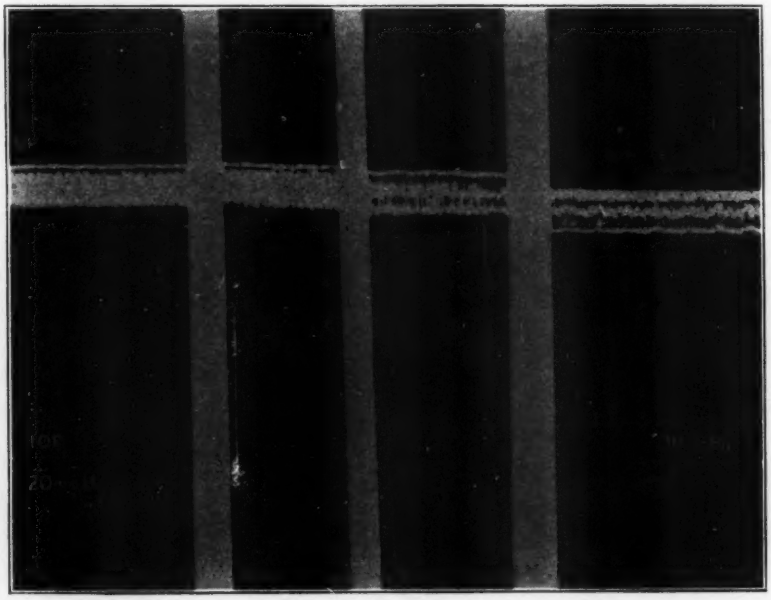

Fig. 17.—The rise of intra-ocular pressure on decreasing the concentration of crystalloids in the perfusing blood by mixing four-fifths blood and one-fifth distilled water.

rounding medium, any variation that it undergoes within the limits compatible with life can be of no effective importance. With regard to the aqueous humor, its volume can be increased or diminished by the osmotic abstraction or addition of fluid, and this can readily be shown experimentally (fig. 16). The depletion of fluid can be brought about by an injection of a substance of high osmotic value into the blood stream, such as a concentrated solution of sodium chloride. Under these conditions, the eye shares the general depletion of all the tissues of the body, and its pressure falls much in the same way as does the pressure in the cranial cavity, as was described by Weed and his co-workers in 1919. If, on the other hand, the reverse conditions are brought about by an injection of a hypotonic solution into the blood stream, a general water-logging of the tissues occurs, and the intra-

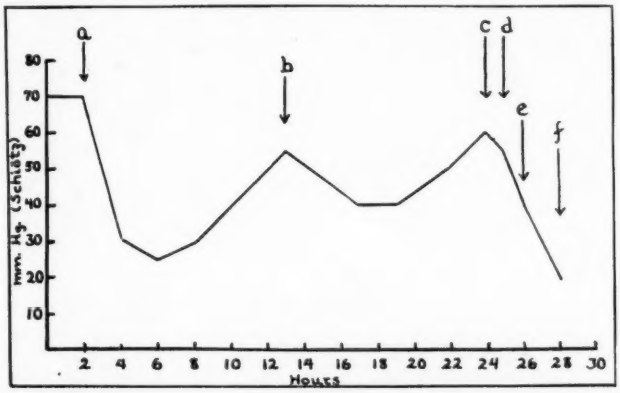

Fig. 18.—Reduction of the intra-ocular pressure with intravenous injection of 20 per cent saline in acute glaucoma: (*a*) 30 cc. of 30 per cent saline; (*b*) 20 cc. of 30 per cent saline; (*c*) 1 per cent physostigmine; (*d*) 35 cc. of 30 per cent saline; (*e*) 1 per cent physostigmine; (*f*) trephine operation.

ocular pressure rises (fig. 17). It is probable that these changes are not of great importance in clinical conditions; but they may be made use of therapeutically, for in cases of acute glaucoma the intra-ocular pressure may frequently be readily lowered by an intravenous injection of concentrated salt solution (fig. 18).

With regard to changes of volume in the vitreous body, I shall consider for a moment the conditions that determine the processes of swelling and shrinkage in gels. It will be remembered that proteins are capable of acting in solution in some conditions as acids and in others as bases, depending on whether the COOH group or the NH_2 group is the more effective. The protein ion may thus carry a negative charge at one moment and a positive charge at another, so that there is always an intermediate point at which these two tendencies are neutral-

ized. Under most conditions, therefore, isolated protein ions react stoichiometrically with acids or alkalis forming highly dissociable metal proteinates or protein-acid salts, but at the iso-electric point the protein exists practically in a non-iogenic condition in which it is able to form neither metal proteinate nor protein-acid salt. It follows that at this point ionization, conductivity, osmotic pressure and turgidity (or swelling) are at a minimum, while here also the protein is most unstable and is, therefore, most easily precipitated from solution. Consequently, when a solution of protein is placed in solutions of varying reactions, all these properties will be found to vary, and a solution of a definite p_H will be found at which they are at a minimum, on each side of which their values increase progressively.

In order to determine the position of the iso-electric point of the vitreous body, I experimented on the vitreous derived from the eyes of animals, subjecting it to various conditions of acidity and alkalinity. Figure 19 shows the result of these experiments; the iso-electric point is in the region of p_H 4 or 5, with the result that in the region of the normal reaction the vitreous swells with an increase of alkalinity and shrinks with an increase of acidity. Figure 20, which shows these changes when the pure proteins of the vitreous body are experimented on, shows a somewhat similar variation; it shows, moreover, that these changes diminish when the concentration of salts has increased. It appears, then, that the vitreous swells in alkalis and shrinks in acids, and one would expect that these changes should be evident as changes in the pressure of the eye. If one experiments on animals in which artificial perfusion of the eye was used, which I have already illustrated, one finds that these results can be obtained, for when the eye is perfused with an alkaline fluid the tension rises, and when it is perfused with an acid fluid the tension falls (figs. 21 and 22).

The question as to what extent these changes enter into the etiology of glaucoma is of great interest. One would expect to find that if the action of the blood or the ocular media became more alkaline, or if the salt content were diminished, the tension in the eye would rise; and conversely, that if the reaction became more acid, or the salt concentration increased, the tension in the eye would diminish. I do not think there is much doubt that the latter set of circumstances are effective in the acidosis of diabetic glaucoma, in which condition a soft eye is frequently seen clinically; but whether so simple a matter can explain the causation of cases of chronic glaucoma is a different question. It must be admitted that the clinical evidence is very strong that in many cases of glaucoma swelling of the vitreous body is of primary importance. The shallow anterior chamber, the obvious pushing forward of the lens and iris, the thinning of the choroid and compression of the retina, combined with the fact that in a fair percentage of cases the angle of

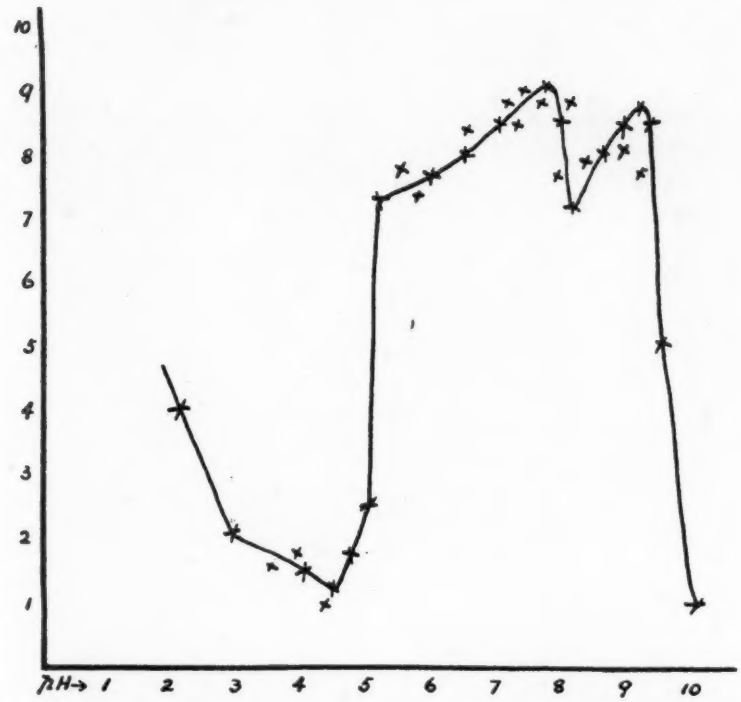

Fig. 19.—The turgescence of the vitreous gel in acids and alkalis. The ordinates denote the final volume of 10 cc. vitreous; the abscissae, p_H values.

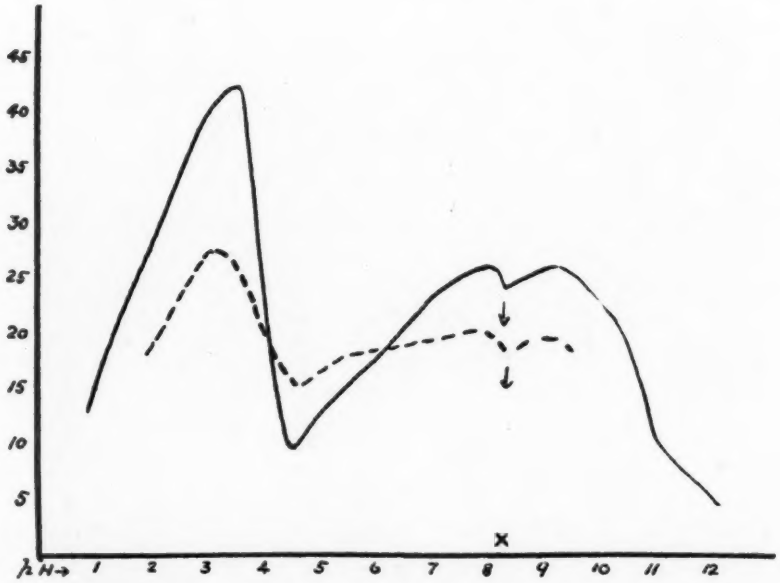

Fig. 20.—The turgescence of the pure proteins of the vitreous in acids and alkalis. The ordinates denote increase of weight in multiples of the original weight; the abscissae, p_H values. The unbroken line represents the variation of the proteins dialyzed salt-free; the broken line, the protein in 15 per cent saline.

Fig. 21.—The rise of intra-ocular pressure in the perfused eye. The alkalinity of the blood is raised to p_H 8 by sodium hydroxide.

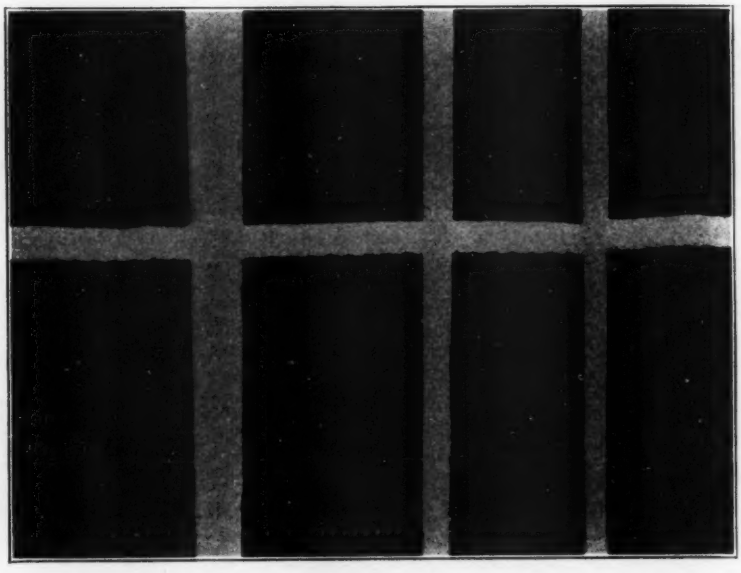

Fig. 22.—The fall of intra-ocular pressure in the perfused eye when the reaction of the blood is changed to p_H 6 by the addition of lactic acid.

the anterior chamber seems to be free from pathologic involvement, point to this conclusion. If a damming back of fluid at the filtration channels were the primary cause, one would look for a deep anterior chamber, owing to the retention of the aqueous humor. The evidence so far at hand, however, would suggest that this swelling of the vitreous is not due to simple changes in the reaction of the blood or in its concentration of salts. There are few bodily phenomena that are so well protected as the maintenance of a constant reaction in the blood, and one does not expect it to vary to an appreciable extent; nor does one find that it does on examining cases of glaucoma clinically. There is a second possibility, however, which, I think, is of much more importance. The integrity of the vitreous body as a gel is maintained by some type of metabolic activity, but the nature of this activity is at present unknown; presumably it is some type of autoxidation mechanism of a nature akin to that which occurs in the lens. It would seem probable that if this mechanism were thrown out of gear, local changes would occur which would alter the interrelation between the various protein constituents of the vitreous, and which would almost certainly produce changes such as swelling and shrinkage. In any case, the elucidation of this possibility is of the greatest moment, and it would seem that the study of the metabolism of the vitreous is one of the most urgent problems to which ophthalmologic research may be directed at the present time.

I think it is probable that it will be found that the etiology of glaucoma is twofold. There is no doubt that many cases are vascular in origin and are produced by an acute vascular crisis in the eye. This would involve a dilatation and an increased permeability of the minute vessels, with an upset of the equilibrium between the intra-ocular fluids and the blood plasma. This may be dependent on general causes—toxemic, infective, dietetic, hormonal; it may be related to a derangement of the sympathetic nervous system, or, on the other hand, experimental evidence is at present accumulating which suggests that it may, to some extent, be due to the local activity of specific substances in the eye, such as histamine. In the second place, there is a swelling of the vitreous body, depending probably on some change in its metabolism. These two may act separately, or they may act together. It may be that, in most cases, the changes in the vitreous depend primarily on vascular changes and a derangement of the first equilibrium; while, on the contrary, it is possible that metabolic disturbances in the vitreous itself may secondarily promote the formation of choline-like bodies in the eye that may produce a vascular crisis in the capillary circulation. Or, more probable still, the one may aid and abet the other, and a vicious circle may be inaugurated and maintained until the eye itself is strangulated.

I would not suggest that the condition of the angle of the anterior chamber is of no importance in the matter. If the filtration channels are free, rises in pressure will be compensated within wide limits, but if they are obstructed the safety-valve mechanism which tends to prevent rises of pressure in the eye by allowing an escape of fluid will be abolished, and rises of pressure will tend to become cumulative. The important point on which I would lay stress is that in the etiology of the disease this is an adjuvant rather than a primary factor.

VITREOUS OPACITIES

In the first part of this paper I have shown that in normal conditions the vitreous body is a gel in a high state of turgescence; as such it possesses an extreme degree of lability. So much is this the case that it can be transformed into a fluid solution by any slight interference with its normal condition or by a very slight trauma. Now this gel-sol transformation is accompanied by the formation of a cloud of minute punctiform opacities and by the separation of the highly gelable residual protein into long films and strands. A similar transformation occurs under the influence of acid or alkaline solutions. In these circumstances the clear mass of gel shrinks up into a small fraction of its original volume; this, of course, is due to a loss of water on a large scale by the individual particles, while the fluid thus lost separates out as a clear solution leaving the shrunken gel mass behind. At the same time, the gel becomes interspersed by an ever-thickening feltwork of white strands and fibers which alternate with gossamer-like membranes floating about in its interior; this represents the deposition of the residual protein in coagel form, and the process goes on until the whole mass becomes turbid and opaque. This behavior is exactly simulated by a gel of gelatin when it is saturated beyond its solubility limit at its isoelectric point, under which circumstances it turns into an opaque suspension.

I have thus arrived at an explanation, or at any rate at a tentative explanation, of the occurrence of one class of vitreous opacities. These may be divided into two kinds: first, exogenous materials, foreign inflammatory products which are derived from the ciliary body, the choroid and the retina in cases of cyclitis, choroiditis and retinitis. The pathology of these is already well understood; they are of the nature of exudates, leukocytes and other inflammatory products, which may remain unchanged for an indefinite time, or may undergo organization, or even may assume frankly purulent characteristics. The second class of opacities is autogenous, derived from the vitreous body itself by its partial or complete breakdown. Such a breakdown can readily be brought about clinically by a derangement of the metabolism of the

vitreous such as would be caused by low grade toxins or by mild inflammatory processes. The strands and diaphanous sheets and membranes seen floating about in a fluid vitreous in a case of chronic cyclitis provide an appearance strikingly similar to that obtained experimentally in the test tube when the proteins, especially the "residual protein," become coagulated out. Such is almost certainly the nature of the so-called "degeneration" of the vitreous in which the mass turns fluid and becomes filled with opacities, as occurs in infective conditions, in senility, in advanced myopia and in detachment of the retina.

RETINAL DETACHMENTS

It is possible to divide retinal detachments into two sharply defined categories. In studying the vitreous gel, it was seen that its protein basis is composed of fine linear micellae, and if these are examined it is found that they tend to arrange themselves in a prevailing unidirectional manner. The consequence is that the vitreous gel itself is not isotropic but contains lines of strain. Thus if gas bubbles are generated in the vitreous in situ by the interaction of hydrochloric acid and sodium bicarbonate, the bubbles are not spherical as would occur if the gel were isotropic down to the smallest spatial units, but they are almost always lens-shaped or arranged lineally, showing that the resistance experienced by the gas in developing is greater at right angles to the surface of the bubble than in it. It will be remembered that the protein basis of the vitreous body is developed from the retina, and in histologic preparations the so-called "fibers" of the vitreous are seen to be directly continuous with Müller's fibers and the internal limiting membrane. It follows that if the vitreous is partly degenerated so that, being partly fluid, these lines of strain are accentuated, they may easily exert a definite pull on the retina with which the residual protein may still retain a connection. If, moreover, an area of the retina is degenerated or diseased, it would seem probable that any such force would be liable to tear it. Suppose, therefore, that one has a fluid vitreous that gives no real support to the retina from within, and that the eye is made the object of a trauma; here are all the materials at hand for causing a detachment of the retina. The protein coagel floating in the vitreous receives a sudden momentum; its adherent base strains on the weakened portion of the retina and readily tears it, making a hole which allows the free vitreous fluid to enter the potential space of the primary optic vesicle; here it collects, extending and perpetuating the detachment. On the other hand, once the hole in the retina is closed, this subretinal fluid, being of the same nature as the intra-ocular fluid, and therefore practically protein-free, will readily be absorbable; in these conditions resolution of the detachment is possible. In these cases the essential precursor and primary

factor in the etiology of a detachment of the retina is a change in the vitreous body involving a shrinkage or a partial liquefaction of the gel mass and the segregation in it of its colloid basis into coagel formations which determine lines of force. Formed by the retina and associated with it embryologically, it will be natural for this colloid material to adhere to any portion of this tissue which shows pathologic change, and on the receipt of a trauma to produce a tear in it, thus allowing the vitreous fluid to gather underneath the retina and detach it. On the other hand, if no localized area of weakness in the retina exists and if no adhesions of the colloid basis of the vitreous body to the retina are formed, a second and totally different type of detachment may occur. Such a detachment can most easily be accounted for by postulating some cause that produces a dilatation at the capillaries of the choriocapillaris; it may be one of many things: trauma, tumor or choroiditis—anything that dilates the capillaries and allows a transudation of colloid-rich fluid from them. In these circumstances, if the vitreous is degenerated and fluid and consequently fails to provide a resilient mass on the inner surface of the retina, this membrane, being unsupported, will allow fluid to collect beneath itself. Once collected in this space, it will of necessity detach the retina; further, this fluid will be gotten rid of only with difficulty, since it is surrounded by membranes relatively impermeable to colloids. The fact that it does contain albumin in this way ensures its permanent retention, for since the protein material cannot escape, considerations of osmotic pressure involve the necessity that the water remain there too.

A final word as to cataract. This disease, too, is simply an exercise in physicochemistry. In the metabolism of the lens two sets of circumstances must be taken into consideration: first, the equilibrium between the intra-ocular fluid and the lens, and second, the autoxidation system of the lens. The integrity of functioning of the former depends partly on conditions in the ocular fluid and in the lens itself, and partly on the capsule which forms the separating membrane. The effects of a disruption of the hydrostatic component of this equilibrium are seen in their most dramatic form in the formation of a traumatic cataract when the capsule is ruptured. The effects of a disruption of the osmotic balance is seen almost as dramatically in diabetes when fluid tends to enter the lens. If the process is mild, the lens swells and is deformed, its curvature and refractive powers are increased, and the acute myopia of diabetes results; if the process is more active, actual droplets of fluid are formed underneath the capsule, and eventually a typical diabetic cataract results. Again, if the efficiency of the membrane is impaired by radiant energy, the abnormal interchange that is thus allowed may, with a direct denaturation of the proteins in the lens, combine to bring about a radiational cataract. The internal autoxidation system forms

a mechanism as important, if not more so, for a disappearance or a diminution of its active components—the β-crystallin and the glutathione —is a constant accompaniment of the development of opacities. Ignorance of the matter is still profound; but it is known that the development of cataract—that is, the denaturation, hydrolysis and coagulation of the proteins of the lens—is a purely physicochemical problem, and it seems almost certain that the essential opacities of the cornea are fundamentally of the same nature.

It is well within the bounds of possibility that all these conditions will before long be explicable on a physicochemical basis, and it may not perhaps be an impossible dream that all of them should eventually be curable or preventable by physicochemical means. But at the present time, one can neither cure nor prevent a single one of them. With regard to some, it is true, inklings have been obtained of possible paths that may lead one far along the road, and here and there one or two sign-posts have been erected leading no one knows whither. But on the whole, the work—work largely in the dark—has progressed to a stage in which the darkness is relieved only by hopes and aspirations, and not within one's lifetime will all of these hopes and aspirations, be realized. In the past the diseases of the eye have been considered almost entirely from the point of view of anatomic pathology—a most useful, and, indeed, a necessary stage in the evolution of knowledge. But hitherto there has been a tendency perhaps to make too much of the structural ruins which disease has left behind, and the deeper and more subtle nature of the initial defect has been overlooked. Further advances in ophthalmology, if they are to be on the grand scale, and if they are to be effective, must, I am convinced, come through the study of biochemistry and physiology.

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CHIASMAL SYMPTOMS IN INTRACRANIAL TUMORS *

LOYAL DAVIS, PH.D., M.D.
Associate Professor in Surgery, Northwestern University Medical School
CHICAGO

Progress in the surgical treatment for intracranial tumors has depended on improvement of surgical technic, diagnosis and a preoperative knowledge of the probable pathologic changes of the lesion to be encountered. With the advent of surgical anesthesia and cleanliness, all operations were thought possible. Yet it has been a long way from the early exploratory trepanations of Jaboulay and the first successful removal of a meningioma by Keen (1888) to present day methods of craniocerebral surgery. Today such an operation is characterized by attention to the meticulous details of an osteoplastic craniotomy, preferably performed under local anesthesia, with complete hemostasis, particularly during the removal of the tumor, replacement of the bone flap, and an accurate closure of the wound, without drainage, designed to leave an invisible scar.

Most neurologic surgeons have come to depend on several devices to accomplish these ends. The brain is sponged with cotton pledgets wet in physiologic solution of sodium chloride instead of with dry gauze; silver clips are used instead of ligatures; bits of muscle are used as stamp implantations; an accurate tracing of the blood pressure is taken throughout the operation; silk sutures are used for closure of the wound layer by layer; a suction apparatus is employed not only to keep the wound clear of fluids and blood, for the emptying of cysts and for the removal of soft gelatinous tumors but to collect the blood lost during the operation for reinfusion into the patient if the emergency arises, and finally an electrosurgical unit is at hand to aid in the complete removal of relatively inaccessible vascular tumors or in the resection of all or part of a lobe of the brain overlying or involved by tumor tissue. These technical matters alone may seem trifling, yet they contribute to the entire picture of good neurologic surgery.

Equally important are matters pertaining to diagnosis. The time has passed when headaches, papilledema and vomiting must be awaited before the diagnosis of an intracranial tumor may be made. Though no instrumental or laboratory aid can equal in importance an exact,

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* Read before the Section on Ophthalmology at the Eighty-Second Session of the American Medical Association, Philadelphia, June, 1931.

detailed history of the symptoms in chronological order and the careful evaluation of all physical signs, several important diagnostic methods are available. Daily one is dependent on the ophthalmoscope and the perimeter to aid in establishing a diagnosis. Improvement in roentgenography enables one to confirm a clinical diagnosis of the nature and precise situation of the lesion by the demonstration of shadows of the tumor or of bony changes and to lateralize the tumor by the pineal shift or by ventriculography.

Of equal if not greater importance than these matters is the microscopic verification and classification of the tumors exposed and a correlation of the pathologic picture with the clinical course of the patient before and after operation. Today the neurologic surgeon must be able to foretell not only the precise situation of the lesion, but its probable pathologic character as well. So it is the present ambition to concentrate on one type of tumor after another in a given location rather than to generalize about all intracranial tumors. Though they have many characteristics in common, the dissimilar behavior of the various tumors is the important matter. There is an increasing number of lesions that can be visualized beforehand with a fair degree of accuracy, and so the surgeon is becoming better prepared to attack each problem with greater confidence.

This knowledge of pathologic matters introduces another important element. The results of a series of operations for a given type of tumor in a given location provide a score for the surgeon to work against. Therefore, there is a constant aim to better his record on the basis of a lower operative mortality and a longer and more livable period of survival following operation.

The intracranial tumors under consideration in this article are those in which the diagnosis rested primarily on changes in the fundus of the eye or in the visual fields. The cases that may have shown identical changes in the fields or fundus as part of a large number of other symptoms of unmistakable localizing value are not included. For convenience in discussing the tumors they may be grouped into the following syndromes, which are characterized by the symptoms which the patients presented: (1) homolateral optic atrophy, contralateral papilledema, anosmia; (2) bitemporal hemianopia and optic atrophy in a middle-aged person with a normal sella turcica; (3) unilateral exophthalmos and optic atrophy; (4) indefinite defects in the fields, optic atrophy and rapid progressive loss of visual acuity; (5) indefinite defects in the fields, papilledema and rapid progressive loss of visual acuity.

SYNDROME OF HOMOLATERAL OPTIC ATROPHY, CONTRALATERAL PAPILLEDEMA AND ANOSMIA

The most common intracranial tumor, which produces the striking syndrome of homolateral optic atrophy, contralateral papilledema and anosmia, is one that arises from the meninges on the floor of the anterior fossa of the skull. Usually, but not invariably, it is situated in the median line and has been designated as a meningioma of the olfactory groove. Foster Kennedy called attention to this syndrome in 1911 in an article entitled "Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobe." In the Macewen Lecture of 1927, Cushing discussed these tumors and emphasized more clearly their clinical significance.

They produce anosmia and optic atrophy by direct pressure at the site of their origin. Contralateral papilledema is produced by the general increase of intracranial pressure. As they increase in size they may come to extend into both frontal fossae, push up the frontal lobes and produce obscure mental symptoms. In addition, slight weakness in the facial musculature on the side opposite the optic atrophy may be present.

The tendency for meningiomas to produce changes in the adjacent bone of the skull is well known. It is not uncommon to see thickening of the floor of the skull with this type of meningioma, though the bony changes are more striking with meningiomas on the surface of the cortex.

A tumor of this type was encountered in a middle-aged woman who was not a particularly favorable surgical risk. Her story is given in the report of case 1.

CASE 1.—*Homolateral optic atrophy. Contralateral papilledema. Anosmia. Mental hebetude. First stage, transfrontal osteoplastic flap; second stage, removal of meningioma of the olfactory groove. Recovery.*

History.—C. C., a woman, aged 51, referred by Dr. F. C. Warne of Chicago on July 20, 1928, consulted an ophthalmologist in 1920 who diagnosed optic atrophy in the right eye. Nothing further was done about this until, in 1927, she became listless, disinterested and unable to carry on her housework. She slept most of the time. There was no complaint of headache or vomiting.

Examination.—The right pupil reacted slightly to light, but the left reacted promptly. Vision was lost entirely in the right field and was diminished in the left, with greater loss in the temporal side. There was a complete primary optic atrophy in the right eye. The retinal veins of the left eye were engorged and there was papilledema. There was a bilateral loss of the olfactory sense. Movements of the left hand were clumsier than those of the right. The left upper extremity fell away when held extended. Deep tendon reflexes in the left upper extremity were greater than on the right. A roentgenogram of the skull showed a thickening of the floor of the right frontal fossa and an indistinct outline of the clinoid processes of the sella turcica.

A preoperative diagnosis was made of a meningioma lying in the right frontal fossa. The syndrome of unilateral optic atrophy, anosmia, contralateral papilledema and mental symptoms made it appear likely that the tumor originated from the olfactory groove.

Operation.—On August 2, a right transfrontal osteoplastic flap was turned down under local anesthesia. There was considerable bleeding from the bone and scalp which was difficult to control. The flap was therefore replaced without opening the dura mater, and a second stage operation was decided on.

On August 7, the tumor was exposed. It proved to be a large meningioma which occupied the right frontal fossa and extended beyond the median line to the left. It was attached about the olfactory groove. The frontal lobe was retracted upward, and it was obvious that the tumor could not be tilted out of its bed by traction sutures. With the electrosurgical unit the tumor was incised and then

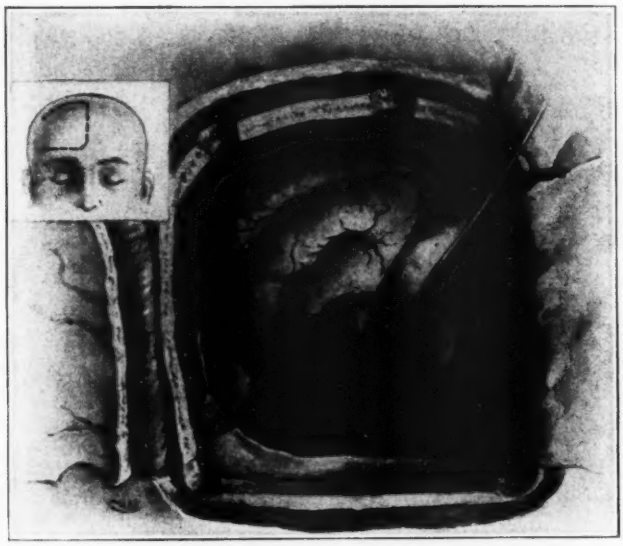

Fig. 1.—Elaboration of operative sketch which shows the location of the olfactory groove meningioma in case 1.

the inside was "looped" out with the coagulating current (fig. 1). This was continued until the tumor had been removed entirely from the floor of the fossa. Successive attempts to tilt the capsula of the tumor by silk sutures met with failure. The tumor was therefore removed in "scallops" down to its attachment, and this was sprayed with the current to destroy any tumor tissue that might remain.

Course.—The patient made a good recovery and was discharged from the hospital on September 13 (fig. 2). A year later she was attending to her household duties and was in excellent condition. The visual field on the left side increased, but the vision in the right eye remained unchanged. Microscopic examination proved the tumor to be a typical meningioma (fig. 3).

This, then, was a patient in middle life in whom there had developed a bilateral anosmia, of which she was unaware, blindness in the right

eye with complete optic atrophy, mental symptoms and involvement of one upper extremity because of an increase in intracranial tension. The latter had only recently produced the first evidences of a beginning contralateral papilledema.

It has been the aim to remove meningiomas intact at one operation, but even when they are situated more favorably this may prove to be a difficult proceeding because of the extreme vascularity of the tumor, its attachments and the surrounding tissues. Without extreme care irreparable trauma to the overlying frontal lobe and uncontrollable hemorrhage may follow an attempt to remove a tumor in such a location.

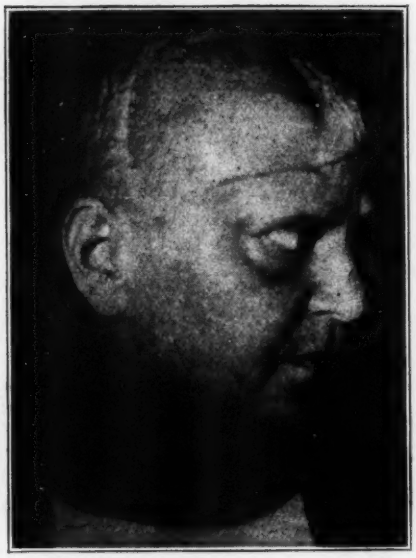

Fig. 2 (case 1).—Photograph of patient on discharge from the hospital after the removal of an olfactory groove meningioma.

It is under such circumstances that the use of an electrosurgical unit has proved so valuable. The operation is based on the application of the well known principle of an intracapsular enucleation such as has been used in the past for the removal of acoustic neurinomas. In other words, there is now at the surgeon's command a method by which a relatively inaccessible, highly vascular tumor, which previously would most certainly have proved inoperable, may be removed in its entirety. Though more accessible, meningiomas have been removed in the past with success; nevertheless they are now approached with less fear of the necessity of multiple operations or of transfusions or reinfusions of blood as life-saving measures.

SYNDROME OF BITEMPORAL HEMIANOPIA AND OPTIC ATROPHY WITH A NORMAL SELLA TURCICA

Recently Cushing called attention to this group of symptoms under the title of the "Chiasmal Syndrome." Among the possible lesions that

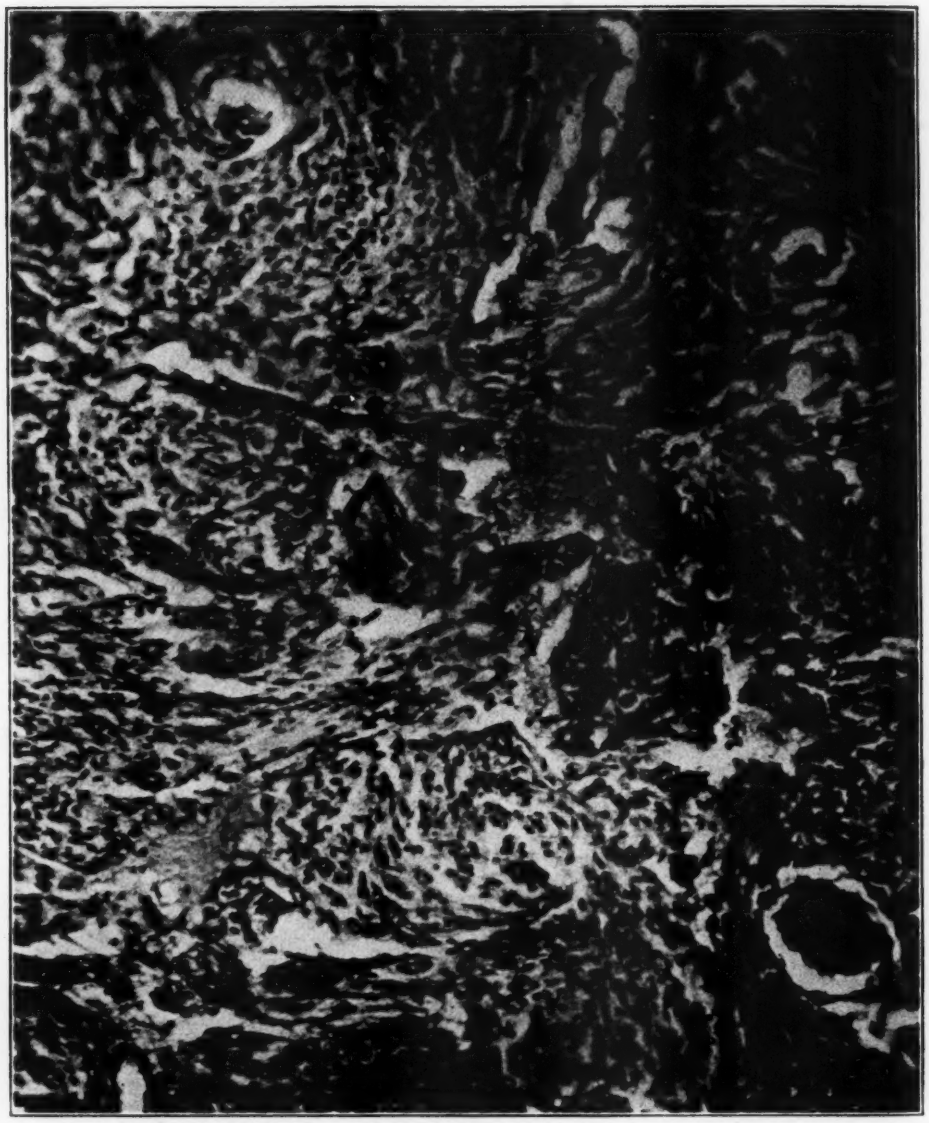

Fig. 3 (case 1).—Section of meningioma. Hematoxylin eosin stain; × 80.

may produce these symptoms are an aneurysm, a primary glioma of the optic chiasm or a congenital suprasellar cyst that has failed to produce symptoms earlier in life and whose walls do not show evidences of calcification in a roentgenogram. A far more common lesion that may

be responsible for this syndrome is a small midline tumor which originates from the meninges over the sulcus chiasmatis or tuberculum sellae.

The major portion of such a tumor may lie beneath the chiasm and raise it upward (fig. 4). The tumors may grow to such a size that their surgical removal is impossible. Moreover, the syndrome just described may become obscured by the effects of hydrocephalus, by involvement of the hypothalamic centers, by evidences of dyspituitarism

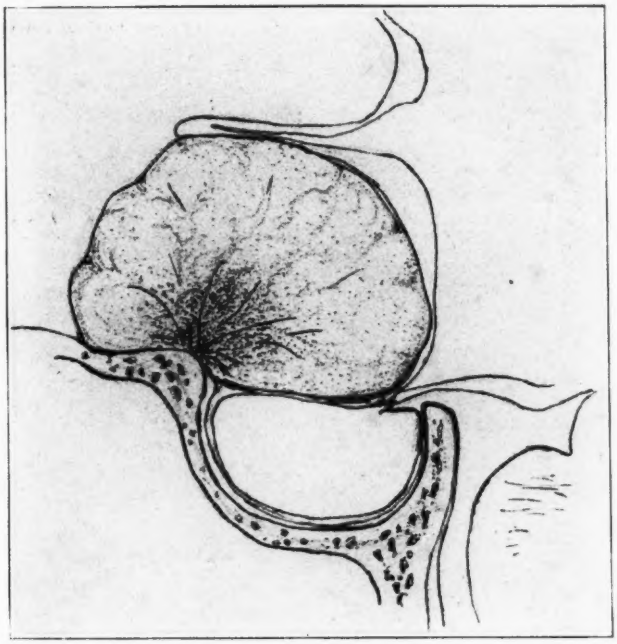

Fig. 4.—Diagrammatic sketch showing site of origin of a suprasellar meningioma (after Cushing).

and by absorption of the sella turcica by pressure. That the tumors are difficult to treat surgically may be well illustrated by the following example:

CASE 2.—*Bitemporal hemianopia. Optic atrophy. Normal sella turcica. First stage, right frontal osteoplastic flap; second stage, removal of suprasellar meningioma. Postoperative hypothalamic symptoms. Recovery.*

History.—H. N., a woman, aged 39, referred by Drs. T. T. Stone and H. S. Gradle on Aug. 15, 1930, had noted that her lateral vision was poor in 1928. She consulted an oculist, who gave her glasses for astigmatism. Her vision continued to fail gradually, and in November, 1929, she consulted an ophthalmologist. Her skull was examined roentgenologically, and some disease of the sinuses was noted. In addition to treatment for this condition, she had five teeth extracted. There

was temporary improvement in her vision. She had only one other complaint, a low frontal headache which extended down over both maxillae like a masquerade mask.

Examination.—No symptoms of disturbance of function of the glands of internal secretion were present. There was a bitemporal defect of the field, which was somewhat irregular and extended more sharply to the midline on the right than on the left (fig. 5). The pupils reacted sluggishly to light. There was a bilateral optic atrophy, but it was more pronounced on the right. There was no evidence of a previous papilledema. Roentgenograms of the skull showed a normal sella turcica (fig. 6).

A preoperative diagnosis was made of a suprasellar meningioma or of a cyst of the craniopharyngeal pouch. I was influenced to make the latter diagnosis because I had recently removed such a tumor which had produced the same syndrome.

Operation.—On September 10, a right transfrontal osteoplastic flap was reflected, and a rose-colored, coarsely granular tumor was found to occupy the space

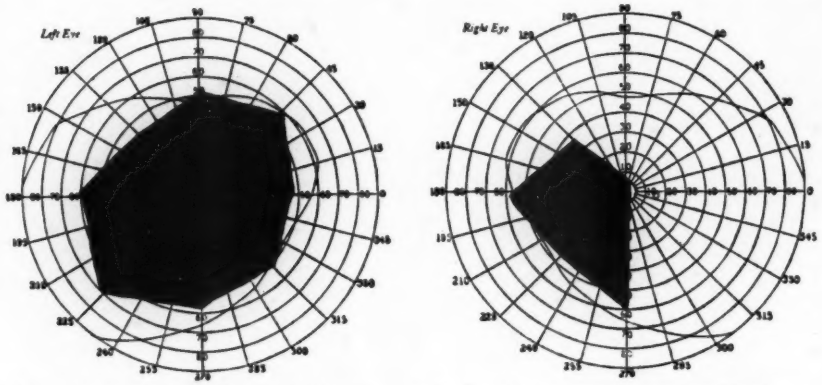

Fig. 5 (case 2).—Visual fields of a patient with a suprasellar meningioma.

between the two optic nerves. The right nerve was flattened and extended posteriorly beneath the flattened optic chiasm, which was pushed upward and backward (fig. 7).

It was impossible to tilt this mass out of its position without first reducing its size. As I attempted to "loop" the tumor out from between the optic nerves with the electrosurgical unit, a short circuit destroyed the instrument. I decided, therefore, to perform a second operation.

Because of a suggestive discharge from the wound which was sterile I waited until September 24 before I attempted to remove the tumor. The flap was elevated and the mass was removed in "scallops" with the electrosurgical loop. As it was thus reduced in volume it could be tilted forward from beneath the chiasm. It was removed from its attachment in its entirety. As I attempted to coagulate the site of the attachment a brisk hemorrhage occurred, which had to be controlled by a muscle stamp.

Postoperative Course.—The patient did well for about ten days. Then a temperature developed that reached 105 F. At the same time the intake and output of fluids ranged around 12,000 cc. These symptoms persisted for six days. A solution of pituitary injected hypodermically controlled the intake of fluids until it grad-

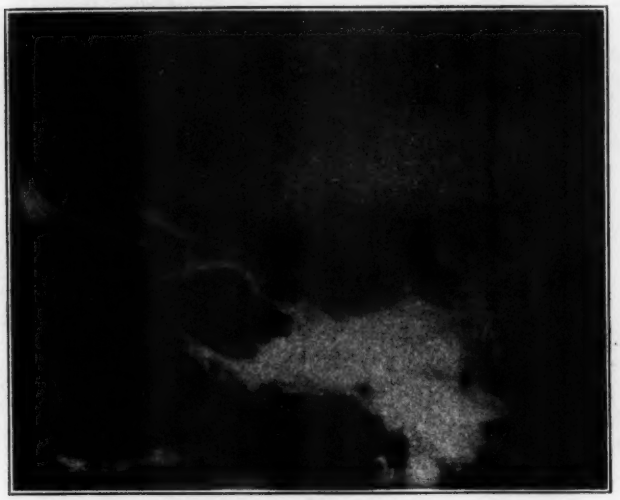

Fig. 6 (case 2).—Roentgenogram of skull of a patient with a suprasellar meningioma. The normal appearance of the sella turcica is shown.

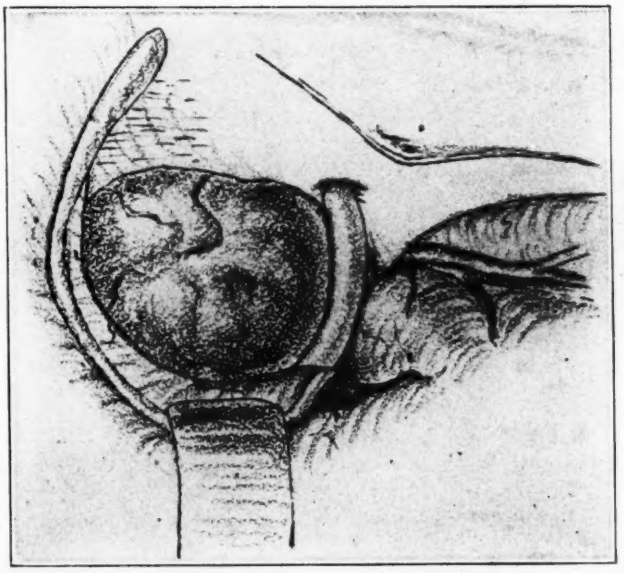

Fig. 7 (case 2).—Elaboration of operative sketch showing the relation of the suprasellar meningioma to the right olfactory and optic nerves.

ually returned to normal. Simultaneously the temperature became normal. The patient improved slowly and was out of bed in a chair a week later. Then rigidity of the neck developed, her temperature rose to 101 F. (rectal) and she appeared to have meningitis. The spinal fluid was under pressure and contained 660 cells, of all of which were lymphocytes. The cultures and smears were negative. This condition continued for three weeks. At no time were cultures positive. The rigidity finally disappeared two weeks later. The highest cell count was 450, and only lymphocytes were present. The fluid again gave negative results on culture.

Fig. 8 (case 2).—Suprasellar meningioma. Hematoxylin-eosin stain; × 80.

The patient began to improve slowly, and was discharged from the hospital on December 22. She continued to improve. Subjectively, the patient's vision was about the same as it was when she was operated on. Perimetric studies were impossible before she left the hospital.

This proved to be a difficult case in which the actual removal of the tumor was the simplest step. Had circumstances been more fortunate this tumor would have been removed at the first operation.

The injury to the hypothalamus became evident some time after the removal of the tumor. The symptoms were simultaneous in their onset with those which indicated meningeal irritation. It is possible that the polyuria, polydypsia and hyperthermia were the result of a basilar meningitis. It is interesting that at no time was there a positive bacteriologic culture, and that all of the cells present were lymphocytes.

On microscopic section the tumor proved to be a fibroblastic meningioma (fig. 8). It is good surgical practice to remove the dural attachment of the meningiomas with the tumor, and this is done when the tumor is situated more favorably. In this instance, coagulation of the cells which may have been left behind in the adherent dura should suffice to prevent a recurrence. This is a more serious problem in the more cellular types of meningiomas that are encountered elsewhere in the intracranial cavity.

If the suprasellar meningiomas were the only tumors that produced bitemporal hemianopia and optic atrophy in a middle-aged person with a normal sella turcica, one's diagnostic worries might be less acute. They are imitated, however, by tumors of the craniopharyngeal pouch which remain quiescent until adult life, and which give no evidence of their presence by roentgen shadows of calcification, which are pathognomonic of their presence.

The recital of an experience with the latter type of tumor strikingly emphasizes that the relief from pressure of the tumor on the optic nerves and chiasm may be followed by a restoration of normal visual fields and acuity.

CASE 3.—*Headaches. Loss of vision. Right transfrontal osteoplastic flap. Removal of tumor of craniopharyngeal pouch. Recovery.*

History.—S. S., a man, aged 32, referred by Drs. T. T. Stone and H. S. Gradle on March 10, 1930, had been well until six months previous to his entrance to the hospital. At that time frontal headaches developed which became progressively more frequent and severe until six weeks before his admission. At about the same time he noted that even perception of light was gone in his left eye. He was fitted with glasses. A week later he had a severe bitemporal headache, and this was followed by a return of vision in the left eye, but a gradual loss of vision in the right eye had been progressive. He was unable to read small newspaper type.

Examination.—There were no physical signs except a sharply defined bitemporal hemianopia (fig. 9). The optic disks were slightly pale, but this sign was so indefinite that it was disputed by several competent observers. The sella turcica was regarded as normal, although attention was called to two minute shadows within the cavity (fig. 10). The basal metabolic rate was normal. There was no polyuria, polydypsia or other secondary hypopituitary symptom.

A preoperative diagnosis of a suprasellar meningioma was made.

Operation.—On March 27, a right transfrontal osteoplastic craniotomy was performed without any surgical difficulties. The frontal lobe was retracted, and a blue-domed structure was seen between the optic nerves, pushing up the chiasm.

Thick, syrupy, yellow fluid was aspirated from this mass, the walls of which then collapsed. Traction on the tumor mass lifted it out of its position. The base of attachment was nodular, yellow and firm. There was no bleeding, and the chiasm fell back under no tension.

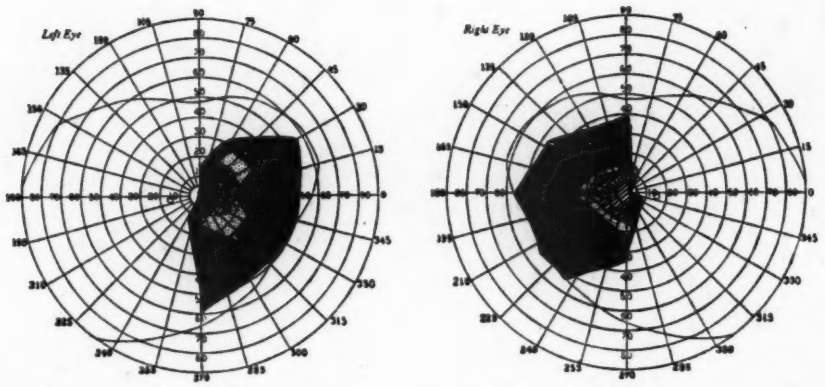

Fig. 9 (case 3).—Visual fields of a patient with a craniopharyngioma, March 27, 1930.

Fig. 10 (case 3).—Very faint shadows of calcification are shown within the sella turcica.

Postoperative Course.—The patient made an excellent recovery, and the next day was conscious of an improvement in vision. On the fourth day, widening of the fields of vision could be demonstrated on the perimeter. His vision continued to improve until at present it is essentially normal (fig. 11).

Microscopic sections showed the presence of hyalinized masses which resembled epithelial pearls. In some areas this material assumed a papillary form lined by epithelial cells. Those cells nearest the hyalinized center were cuboidal, while those in the periphery resembled vesicular squamous cells. The picture was that of a typical adamantinoma or tumor of the craniopharyngeal pouch (figs. 12 and 13).

Cushing has suggested the term "craniopharyngioma" to designate this group of congenital tumors which in the past have been called adamantinomas, tumors of Rathke's pouch, suprasellar cysts and cysts of the craniopharyngeal duct. Since they are of congenital origin they usually give rise to symptoms before adult life. About 80 per cent of these cases show roentgen evidence of the deposit of lime salts in the walls of the tumor (fig. 14). Consequently they may be recognized rather quickly by the roentgenologist.

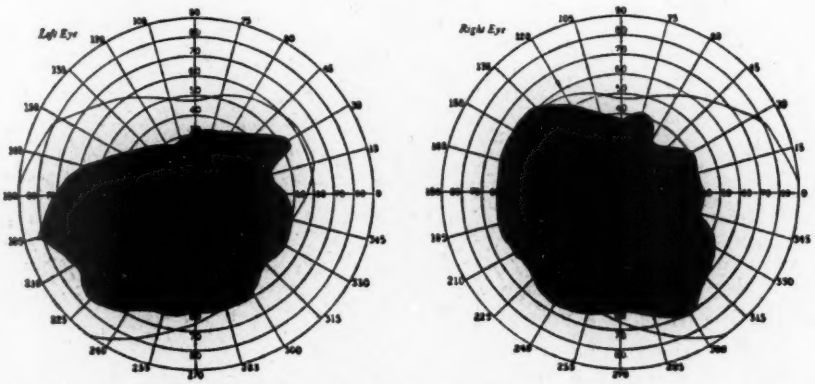

Fig. 11 (case 3).—Visual fields of a patient after the removal of a craniopharyngioma, on Jan. 10, 1931.

If the tumor lies within the sella turcica, it may "balloon out" that structure as does an adenoma of the pituitary gland. On the other hand, it may lie above the sella and produce a secondary hydrocephalus accompanied by a papilledema. Evidence of a secondary hypopituitarism may appear late in life if the tumor has remained quiescent. If it produces symptoms early in life, signs of sexual and skeletal infantilism may be present.

Following the operation in case 3 I realized that I should have been positive in my identification of the calcified flecks that were present within the sella. On the other hand, the patient belonged more accurately within the age limit of the meningioma group. It may be realized that without well marked shadows of calcification, pituitary symptoms or changes in the sella and with a bitemporal hemianopia, it may be impossible to differentiate these tumors from a suprasellar meningioma.

Fig. 12 (case 3).—Craniopharyngioma. Davenport stain: × 60.

Fig. 13 (case 3).—Patient after the removal of the craniopharyngioma. Inconspicuous scar is seen.

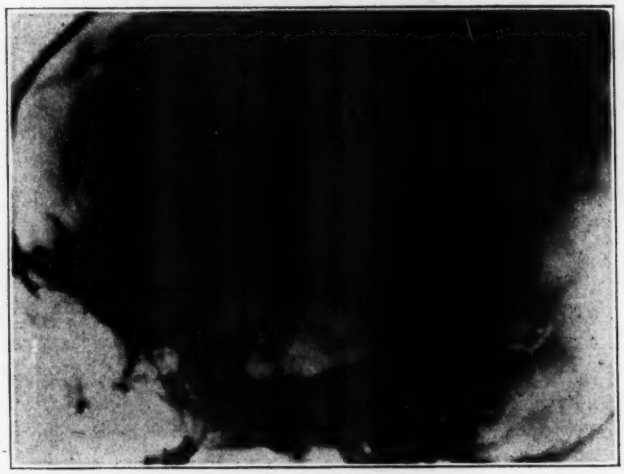

Fig. 14.—Roentgenogram showing typical calcification in a craniopharyngioma.

SYNDROME OF UNILATERAL EXOPHTHALMOS AND OPTIC ATROPHY

It is known that the meningiomas arise from the villous projections of the arachnoid into the dura and the clusters of cells asociated with them. These structures are found along the larger dural sinuses, in the basal meninges, particularly over the tips of the temporal lobes and about the gasserian ganglion. These arachnoid villi act physiologically as filters in the pathway of the cerebrospinal fluid between the subarachnoid spaces and the dural sinuses. Under the stimulus of inflammation or injury the clusters of cells in the arachnoid proliferate and become phagocytic. It is easy to imagine that they may remain permanently hypertrophied and as a result of succeeding stimulations may even assume neoplastic tendencies under circumstances as yet not understood in relation to the genesis of tumor formations. Clusters of arachnoidal cells are found about the sheath of the optic nerve, and tumors arising in that location are not uncommon.

These meningiomas produce a unilateral, painless exophthalmos associated with a homolateral optic atrophy. It is not uncommon for the meningioma to invade the frontal bone and produce a marked deformity. The tumors may also grow posteriorly and produce an obstruction in the pathway of the cerebrospinal fluid. In such instances there may be a contralateral papilledema.

An example of this group of patients who usually consult the ophthalmologist first is given in case 4.

CASE 4.—*Right optic atrophy. Left papilledema. Right exophthalmos and ophthalmoplegia. Headaches and epileptic seizures. Right transfrontal osteoplastic flap. Removal of meningioma. Recovery.*

History.—W. W., a man who entered the hospital on Jan. 26, 1930, had noted a beginning and gradual failure of vision in the right eye as early as 1920. He had had severe headaches which began in the right occipital area and radiated to the temporal region. Several generalized epileptic seizures had occurred since 1923. In 1928, the right eye began to protrude, and the condition progressed rapidly (fig. 15).

Examination.—The patient was completely blind in the right eye. There was marked proptosis of that eyeball, which was immovable. There was right primary optic atrophy with papilledema in the left eye. Movements of this eye were normal. The left side of the face moved less than the right. The olfactory sense was normal. Roentgenograms of the skull showed a thickening of the orbital ethmoidal area on the right. The right frontal sinus was obliterated (fig. 16).

Operation.—On February 17, a right transfrontal osteoplastic flap was reflected under local anesthesia. The scalp was very vascular and the frontal bone very thick. A ventricular puncture lowered the intracranial tension so that the frontal lobe was retracted without opening the dura mater. Soon a reddish-purple mass was exposed on the floor of the anterior fossa, which extended through the orbital plate into the orbit.

With the electrosurgical "loop" electrode this mass was removed piecemeal, slowly and carefully. It was evident that somewhere below its surface I would

encounter the optic nerve. Brisk bleeding occurred from the tumor mass which I could control only by using implants of muscle. The patient's blood pressure fell precipitately, and his general condition was poor. It was decided, therefore, to remove this tumor in multiple stages. The scalp, without the bone flap, was replaced after the dura mater over the temporal bone had been opened.

Course.—The patient did very well following the operation. The exophthalmos receded, and the papilledema subsided. He was allowed to go home until May 1, when he returned for another operative attack on this extensive tumor. The flap was reelevated, and the tumor was removed piecemeal with the electrosurgical loop. The right optic nerve was dissected free from the overlying mass, and the orbital cavity was cleared of tumor as completely as was possible. The growth

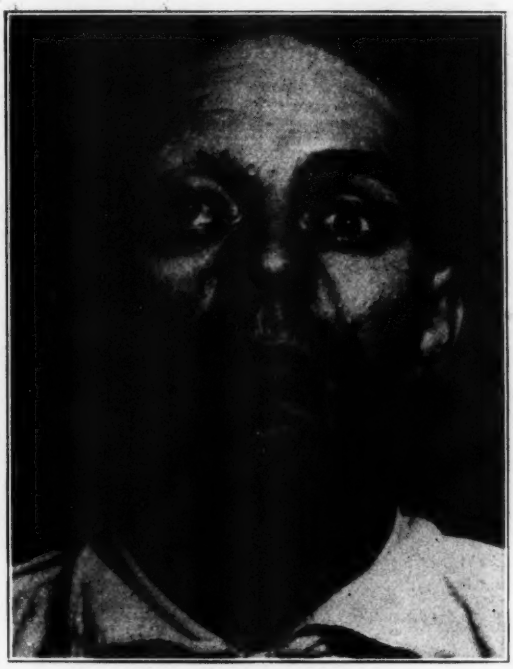

Fig. 15 (case 4).—Unilateral exophthalmos is present.

extended posteriorly and laterally, to occupy the entire right frontal fossa. I felt that some tumor cells were of necessity left behind, and as it was the cellular type of meningioma, I have strong fears that it may recur. The vision in the right eye has not improved, but the papilledema has subsided completely, and the patient has satisfactory vision in the left eye.

I had a similar case in a woman first operated on elsewhere in 1920. At that time a flat tumor was removed from behind the orbit, and it was felt that a complete removal had been accomplished. Five years later, a tumor three times as large was removed from the middle and anterior fossae in the same patient. It was evident that at the first operation the tumor had broken off over the anterior sphenoidal ridge,

which it had straddled in its growth. The patient has been well since that time although she, too, never recovered vision in the atrophied eye.

Carefully taken roentgenograms of the skull will often show the thickening of the floor of the frontal fossa which is produced by these foraminal meningiomas of the cranial nerve. Even without such evidence the unilateral exophthalmos with an optic atrophy should be enough to suggest the presence of such a tumor.

Fig. 16 (case 4).—Roentgenogram of the skull of a patient with a meningioma which extended into the orbit.

SYNDROME OF INDEFINITE DEFECTS OF THE FIELDS, OPTIC ATROPHY AND RAPID LOSS OF VISUAL ACUITY

Gliomas of the optic chiasm produce symptoms that point directly to involvement of that structure. I have encountered three of these tumors in my series of 191 verified intracranial tumors. Their intraorbital characteristics have been discussed in detail by ophthalmologists, particularly by Verhoeff. Martin and Cushing described 7 cases in 1923 and Cushing later, in 1930, referred to a group of 18 cases. It

is apparent that neurologic surgeons have viewed them from their less favorable side, in which the involvement of the hypothalamus and the third ventricle is a matter of millimeters.

While these tumors undoubtedly are more frequent in children, 2 of my patients were in adult life. In all, the course of the progressive loss of vision was rapid. In one there were associated symptoms of dyspituitarism that made the preoperative diagnosis uncertain. In all instances the defects in the fields were bizarre and rapidly progressed to blindness in one or both eyes.

An illustration of this group of tumors is found in the following report of a case:

CASE 5.—*Rapid loss of vision. Polyuria. Drowsiness. Left hemiparesis. Bilateral optic atrophy. Right transfrontal osteoplastic flap. Death.*

History.—K. D., a girl, aged 5, was referred to the hospital by Dr. A. B. Yudelson on Dec. 3, 1926. In February the patient's mother first noticed her loss of vision. The child bumped into objects as she walked about her home. This loss of vision progressed so rapidly that by May she was completely blind in the right eye. She then began to urinate frequently and drank large quantities of water. Her mother also noticed that she slept a great deal during the day. This latter symptom lasted until September. At that time she began to drag her left leg as she walked. Soon it was noted that her left arm and the left side of her face were weak.

Examination.—There was bilateral optic atrophy. The patient was completely blind in the right eye, but could count fingers at a distance of 1 foot (30 cm.) with the left eye. Neither pupil reacted to light. The left side of the face showed definite muscular weakness on emotional expression. The left deep tendon reflexes were greater on the left side than on the right. There were six, macular, light brown, pigmented areas in the skin over the chest and back. These were not tender. Roentgenograms of the skull showed a depression in the bone between the anterior clinoid processes of the sella turcica.

An osteoplastic craniotomy was advised, and a diagnosis was made of a lesion involving the chiasm and extending upward into the midbrain.

Operation.—A right transfrontal osteoplastic flap was made on December 22. A large firm mass that involved the chiasm and extended forward into the optic nerves was found. This tumor so replaced the chiasm and extended backward and upward into the third ventricle that normal anatomic landmarks were obliterated. A section was taken for microscopic study, and the wound was closed.

Course.—The child did fairly well for twenty-four hours after operation. Then a temperature of 107 F. developed which was maintained in spite of all efforts to reduce it until her death on December 24.

Autopsy.—Unfortunately, the brain was sectioned before it had been hardened properly. However, the enormous mass that involved the chiasm could be seen pushing backward and upward into the third ventricle and midbrain. No other cranial nerves were involved (fig. 17). Microscopic sections showed numerous unipolar spongioblasts throughout the tumor. There was also a considerable amount of intercellular fibrous tissue.

This was a desperate case and inoperable. Had I been able to visualize the pathologic condition I should have relied on high voltage roentgen therapy alone. The other cases in the series were more favorable, since the gross pathologic changes were less extensive. One of these patients had a bulbous tumor that involved the right optic nerve and crowded its way into the optic foramen. The chiasm was only partially involved.

In the other patient, who had pronounced symptoms of dyspituitarism that seemed to class his case as an example of a hypophyseal adenoma with Fröhlich's syndrome, the chiasm and the right optic nerve were

Fig. 17 (case 5).—Sagittal section of the brain showing extension of a glioma of the optic chiasm into the third ventricle.

involved. These associated symptoms were undoubtedly due to hypothalamic pressure, because the normal hypophysis could be visualized within the sella. Enlargement of the optic foramina in these cases may often be seen in the roentgenogram. Such changes, however, are always more obvious after verification of the tumor than before the operation has been performed.

The surgical treatment of these tumors does not have a particularly bright outlook. Cushing has reported the removal of a rather well localized tumefied segment of one optic nerve. The patient died within six months, showing evidences of an intracranial extension of the process. In none of my cases would it have been possible or wise to attempt its removal. The greatest danger in the surgical treatment

in these cases is the onset of hyperthermia, which follows the slightest disturbance of the hypothalamic mechanism. Perhaps intensive high voltage roentgen therapy may hold some hope for delaying the progress of these tumors. There has been no marked advance in the symptoms

Fig. 18.—Glioma of the optic nerve. Davenport stain; × 24. There are numerous unipolar spongioblasts in the section.

of one of my patients so treated during a six months' period following operation.

This syndrome of optic atrophy, indefinite defects of the fields or blindness without other localizing signs, may lead to an unfortunate error in surgical judgment. In case 6 there seemed to be additional evi-

dence of changes in the sella turcica, which pointed to a chiasmal lesion. Though this case belongs in the group of tumors in rare situations which produce symptoms of a commonly recognized syndrome, it will serve to emphasize the diagnostic importance of certain facts.

CASE 6.—*Loss of vision. Headaches. Gain in weight. Optic atrophy. Right transfrontal osteoplastic flap. Tumor unverified. Right parieto-occipital osteoplastic flap. Partial removal of meningioma. Left parieto-occipital osteoplastic flap. Further removal of meningioma.*

History.—D. McG., a man, aged 38, referred to the hospital, Oct. 1, 1928, had noticed difficulty in vision as long ago as 1918, when he was in the army. He was fitted with glasses and continued his work as an accountant until 1926, when his sight forced him to do other work. From that time until his entrance into the hospital he was treated for disease of the sinuses and had his glasses changed many times. He suffered from rather severe bitemporal headaches, which had become progressively worse.

Examination.—The results of the physical examination were without note except for the ophthalmologic observations. The right pupil was dilated and did not react to light. The left pupil reacted to light and in accommodation. He was blind in the right eye, and vision in the left eye was 1/10 at 5 feet (152 cm.). The left visual field was so small that it could not be evaluated. The right optic nerve head was pale; the left less so. The retinal veins were somewhat engorged. Papilledema was not present.

I felt that I was dealing with a lesion about the optic chiasm. It was not clear what the pathologic process might be, though I considered a suprasellar meningioma, an adenoma of the hypophysis, a glioma of the chiasm and arachnoiditis.

First Operation.—On October 15, a right transfrontal osteoplastic flap was reflected. The third ventricle was enormously dilated and projected forward over the chiasm. The optic nerves were normal in appearance. The arachnoidal cisterns about the chiasm were normal, and the hypophysis was visualized.

The patient made a good recovery and had some subjective improvement in vision. I could never record an objective improvement. However, he was able to work in his delicatessen store for the next year. During that period several follow-up examinations failed to reveal any new symptoms.

In November, 1929, however, he complained for the first time of hallucinations of lilliputian figures. These were not hemiopic, and they came in attacks. During the attacks he became completely disoriented in his own home. He was not able to determine the general direction of his bathroom or to distinguish left from right.

On November 19, he reentered the hospital. An encephalogram was made on November 20. The ventricles did not fill with air. The subarachnoid spaces over the frontal lobes were dilated. A very small amount of air was found in the subarachnoid spaces over the parietal and none over the occipital lobes. The most striking change, however, was in the skull itself. In the midline of the occipital bone there was thickening of the skull. This extended more to the right than to the left. It was immediately suggestive of the changes in the bone due to a meningioma. The patient wished to enter the hospital after the holidays for an operation (fig. 19).

Operation.—On Jan. 22, 1930, a right occipital osteoplastic flap was reflected. The bone was so vascular and involved in tumor tissue that it had to be removed. Tumor tissue which proved to be a meningioma had grown through the dura mater

into the bone. This mass extended across the sagittal sinus and falx to the left side. On the lateral surface of the right hemisphere it extended down about half way. It extended forward to the postcentral gyrus. This part of the mass was removed with the overlying dura mater to the falx, by the electrosurgical unit. It was necessary to give the patient a reinfusion of blood collected during the operation. He made a good immediate recovery.

Third Operation.—On January, 27, a similar operation was performed on the left side. The bone was not involved so extensively, but had to be removed over the median line. The tumor tissue that extended over the lateral surface of the left hemisphere was removed. Again a reinfusion was necessary. The tumor proved to be extremely vascular and soft. It was obvious that a mass, perhaps

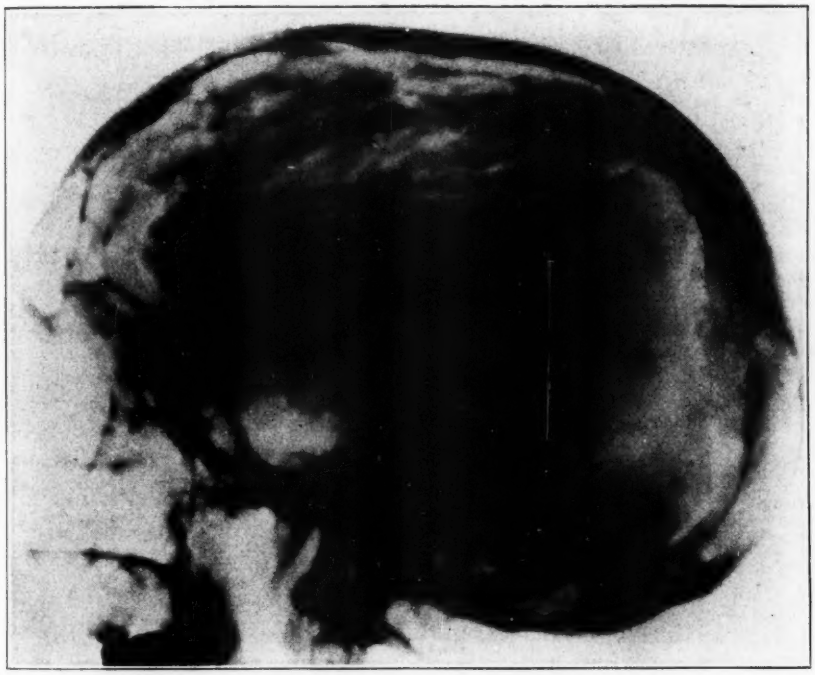

Fig. 19 (case 6).—Roentgenogram of the skull. Note the enormous thickening of the bone in the parieto-occipital area. Air outlines the frontal cerebral subarachnoid spaces.

larger than that which had been removed, was attached to the falx cerebri and extended between the occipital lobes.

Fourth Operation.—On February 3, another attempt was made to remove the remainder of this enormous tumor. It was removed in scallops from the sides of the falx by the electrosurgical unit. The patient's condition became precarious, and the operation was stopped. He recovered more slowly from this procedure, and in spite of transfusions of blood his red blood cell count could not be raised.

Course.—It became obvious that I was dealing with a tumor that had grown beyond the usual surgical limits. Though he was blind I felt that I should try to save his life. It was necessary to give him an opportunity to regain strength before I could go further. Instead of improving, the patient lost ground steadily, and he died on June 15.

This case presents many difficult problems in diagnosis and treatment. One of the important lessons to be learned is that concerning the roentgen appearance of the sella turcica. It appeared to have been destroyed by pressure from without, particularly from above. It did not have the appearance of the "ballooned out" sella produced by an intrasellar adenoma of the hypophysis. In the latter case the floor is usually eroded, and the sella is destroyed by an expanding lesion from within. This difference was recognized before operation. It was attributed, however, to a chiasmal lesion rather than to a dilatation of the third ventricle.

The visual fields were so contracted that no help from them was possible. It would be interesting to have had a record of the change in the visual fields as this lesion progressed.

The next factor that is confusing in considering the case is that such a large tumor did not produce papilledema. Neither did the patient have severe headaches during my observation of him. The lack of intracranial pressure and the normal size of the ventricles may be explained by the enormously dilated subarachnoid spaces over the frontal and parietal lobes.

Perhaps the changes in the skull over the tumor were present before they were recognized. When they were found, the evidence was unmistakable. In the light of the outcome of the case, a review of the films taken on the patient's admission to the hospital does not show any changes that could be definitely diagnosed as thickening due to a tumor.

This tumor should be classified with that group, fortunately small, which produce false localizing symptoms. They are less frequent as additions are made to the general knowledge of diagnostic problems. While I have removed meningiomas attached to the falx cerebri and have ligated the superior longitudinal sinus to accomplish it, the size of this tumor in its particular location exceeded the boundaries of surgical possibilities.

SYNDROME OF INDEFINITE DEFECTS OF THE FIELDS, PAPILLEDEMA AND RAPID LOSS OF VISION

The only difference between the symptoms in the cases that belong to this syndrome and those of the former group rests on the presence of a papilledema instead of an optic atrophy that has not been preceded by a papilledema. This syndrome is distinguished from the others by the rapid development of loss of vision without a defined hemiopic or quadrantic defect of the visual field.

All but one of the cases in this group have been classified in my series as "chiasmal cisternal arachnoiditis" and, what is more important, as "suspected intracranial tumor." While I have microscopic evidence

of the pathologic changes in the arachnoid membrane, I feel that autopsy alone will allow one to state that a tumor is not present. This is my belief in spite of the fact that all patients have shown definite clinical improvement, in some instances as long as three years.

Case 7 is an example of one of these cases that was mistaken at operation for a chiasmal glioma.

CASE 7.—*Headaches. Loss of vision in two weeks. Papilledema. Right transfrontal osteoplastic flap. Recovery.*

History.—In C. B., a boy, aged 7, referred to the hospital on March 5, 1928, by Dr. Thomas Cooper, of Logansport, Ind., a severe generalized headache had suddenly developed on January 23. A few days later, there was some soreness

Fig. 20 (case 7).—Appearance of the patient on the tenth postoperative day.

of the neck. These symptoms then disappeared. On February 5, he again complained of headache and drowsiness. His condition remained about the same for four days. On February 9, he complained of pain above the left eye and blurring of vision in that eye. On February 11, he could not see out of the left eye, and papilledema was present. On February 25, he had pain over the right eye and blurring of vision. By March 1, he could not see with his right eye. His headaches had disappeared.

Examination.—The patient was totally blind. Both pupils were widely dilated. There was a bilateral papilledema with hemorrhages about the disk. The right disk was swollen 5 diopters; the left about 3. The left disk was paler than the right. The sella turcica was normal. A presumptive diagnosis was made of a chiasmal lesion, but no opinion was ventured as to its nature.

Operation.—On March 8, 1928, a right transfrontal osteoplastic flap was reflected. When the subarachnoid space was opened about the chiasm an enormous amount of clear cerebrospinal fluid escaped under pressure. The ventricles were

also dilated. The arachnoid about the chiasm and optic nerves was thickened and opaque. It could be lifted up and removed in sections. The right optic nerve was at least three times its normal size. It was red, and the vessels on the surface were dilated. It was regular in its enlargement. The left nerve was also enlarged but was about one-half the size of the right. The chiasm was large, red and surrounded by arachnoid. The right nerve was pushed into the foramen so that a cuff formed about the opening. The nerves were firm but not unyielding to palpation.

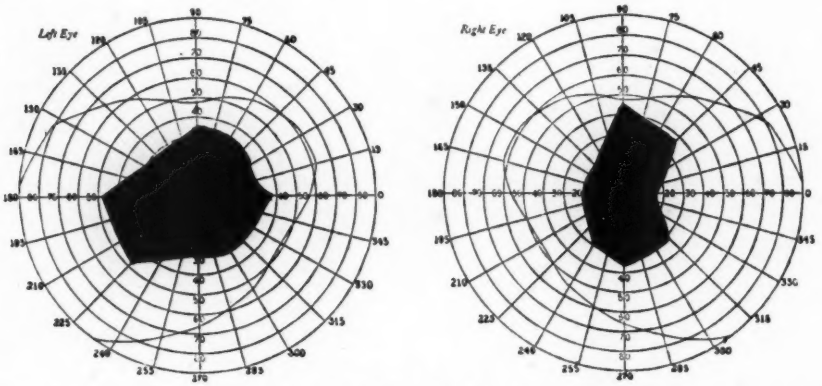

Fig. 21 (case 7).—Defects of the visual fields on April 23, 1928, after operation.

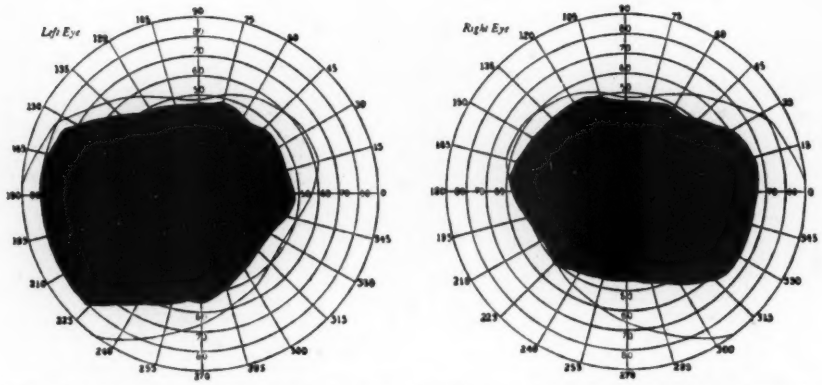

Fig. 22 (case 7).—Defects of the visual fields on Aug. 9, 1928, after operation.

Course.—A piece of arachnoid was saved for microscopic examination although my impression was that I had exposed a glioma of the optic chiasm which extended down both nerves. The course of the development of symptoms was against that view, however. The patient made an excellent postoperative recovery. On his discharge, on March 18, he could get around his room alone, and rough tests showed a definite improvement of vision (fig. 20).

On April 23, 1929, his visual fields showed marked improvement (fig. 21). Visual acuity was 15/200 in the left eye, and he could count fingers at 3 feet (91 cm.) with the right eye. On Aug. 9, 1929, his vision was 20/50 in both eyes; the fields are shown in figure 22. When examined on March 3, 1930, the fields were about the same. The optic nerve heads show an atrophy which has not progressed. He is attending school and has been well.

The arachnoid has been studied in a group of cases presenting symptoms of tumor in which a thickened arachnoid membrane was the only recognizable pathologic sign. I have been able to classify the pathologic changes as inflammatory, fibrotic and hyperplastic. Figure 23 illustrates the microscopic appearance of each of these types of change. This case belongs to the inflammatory group, and these as a rule give the best clinical results. It is well known that an infection of the middle ear may be accompanied by a leptomeningeal reaction in the cerebellopontile angle; that a similar process may occur in the chiasmal arachnoid cisterns is logical. A definite history suggestive of an infectious onset is present in this instance. Cases have been reported in which a sub-

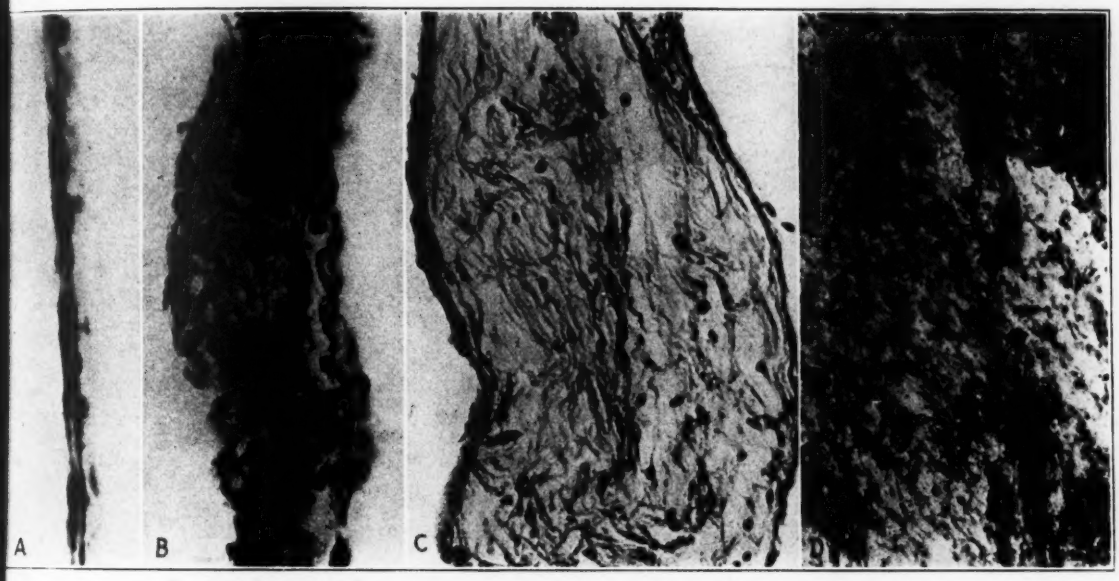

Fig. 23.—*A*, normal arachnoid membrane; *B*, hyperplastic; *C*, fibrous, and *D*, inflammatory arachnoid changes.

temporal decompression alone has been followed by complete subsidence of all symptoms. The recovery of vision in this case was particularly gratifying in view of the impression received at operation. It must be emphasized again that these patients must be suspected of having a tumor until autopsy proves conclusively that none is present. A less rigorous attitude will tend to dull one's diagnostic acumen.

There is no doubt that operations on the accessory nasal sinuses have been followed by improvement in conditions diagnosed as retrobulbar neuritis. The rapid loss of vision makes it imperative, it would seem, to be positive about the condition around the chiasm and optic nerves in such a patient. Neurosurgical procedures have been improved to such an extent that an osteoplastic flap is not particularly dangerous.

Seemingly less drastic operations may often cause a delay with resultant optic atrophy from which recovery is impossible. The important question is to decide whether or not an intracranial operation is indicated from the appearance of the fundi. This decision must be made by the ophthalmologist, whose opportunity comes too late many times.

Again it is necessary to call attention to the occurrence of an intracranial tumor, the location of which must have been somewhat

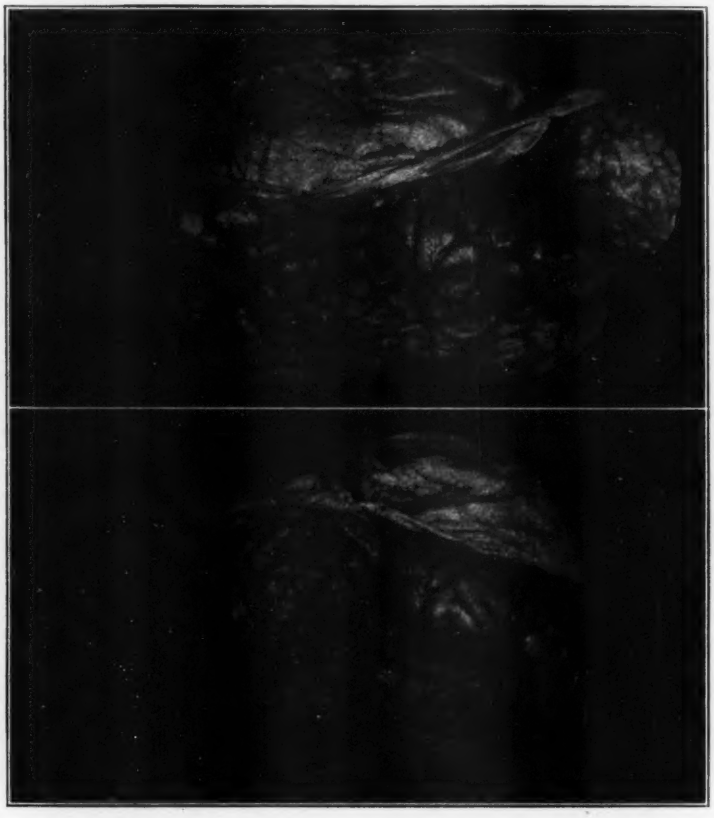

Fig. 24 (case 8).—Appearance of autopsy specimen. Chiasmal symptoms had been produced by this tumor.

unusual. Yet, it gave rise to a train of symptoms that were difficult to evaluate. Moreover, the case illustrates the unfortunate circumstances that commonly attend a mistaken diagnosis of localization in neurologic surgery.

CASE 8.—*Headaches. Loss of vision. Bilateral papilledema. Erosion of sella turcica. Transfrontal osteoplastic flap. Death. At autopsy a meningioma found between the occipital lobes attached to the falx cerebri.*

History.—A. L., a woman, aged 54, referred by Dr. Arthur Metz of Chicago, began to complain of severe headaches accompanied by vertigo and vomiting in

May, 1925. In January, 1926, her local physicians had had her teeth extracted to eradicate foci of infection which they believed were the cause of a hypertension. She was not relieved, and in April, 1926, she underwent two intranasal operations without relief. At that time she began to have a loss of vision, which rapidly progressed to blindness.

Examination.—The patient showed a bilateral papilledema. The right disk was swollen 2 diopters and the left, 4 diopters. There were a few scattered hemorrhages in the retinas about the disk. She was dull mentally and was difficult to examine. She could not detect light in either eye. She showed many symptoms of dyspituitarism and had not had menses for twelve years. The roentgenograms showed erosion of the sella turcica without the characteristics of the "ballooned out" sella commonly seen in a hypophyseal adenoma.

Operation.—On May 5, 1926, a right transfrontal osteoplastic craniotomy was performed under ether anesthesia. Careful exploration of the optic chiasm and sella turcica did not reveal any pathologic changes. The wound was closed, and the patient was in good condition. Twelve hours later, hyperthermia developed, and the patient died.

Autopsy.—A large, spherical, firm, tumor mass was found between the occipital lobes of the brain. The mass rested on the tentorium, but was definitely attached by a pedicle to the posterior portion of the falx cerebri (fig. 24). Microscopic sections proved it to be a meningioma.

It must be remembered that this patient showed no other symptoms besides those referable to the eyes. Unfortunately, I did not know what the visual fields showed earlier in the course of the illness. They might have been of considerable aid in the prevention of a wrong conclusion.

The destroyed sella turcica was similar in appearance to that produced by pressure from a dilated third ventricle. It alone should have set me right had I not allowed the symptoms of dyspituitarism which she showed to outweigh its significance. All the latter may be explained as the result of secondary pressure.

It is apparent that this tumor should have proved comparatively easy to remove. It had a definite pedicle-like attachment to the falx cerebri. It had not produced any change in the thickness or contour of the occipital bone by which its presence might have been surmised.

COMMENT

It is not my purpose to call attention to the similarities of the various lesions about the optic chiasm; rather, it is to emphasize their definite dissimilarities. A normal sella turcica in a patient with bitemporal hemianopia and optic atrophy is the symptom that points to the fact that the lesion is different than the common expanding adenoma of the hypophysis. Constant improvement in neurologic surgery depends on the completeness of the preoperative diagnosis. To be forearmed with a knowledge of the pathologic origin and characteristics of a tumor

as well as with an accurate knowledge of its localization means the difference between success and failure.

Although there are many other symptoms that are necessary to portray the entire clinical picture of these chiasmal lesions, accurate perimetric charts and a satisfactory examination of roentgenograms of the sella turcica are invaluable. No one knows better than the ophthalmologist how difficult it may be to obtain reliable data on the visual fields. However, the neurologic surgeon realizes more fully how important they prove to be in diagnosis. The success or failure of his operative procedure is measured in terms of restoration of the visual fields and acuity. There is no doubt that careful studies of early records of visual fields in these cases will show changes of diagnostic value. These, in turn, will lead to early surgical therapy, and secondary hypothalamic symptoms either before or after operation may be avoided.

With few exceptions, the changes in the skull produced by intracranial tumors have been pointed out by neurologic surgeons. The change in the sella turcica produced by an expanding lesion such as a hypophyseal adenoma has been characterized by the term "ballooned out." The clinoid processes are pushed back and thinned. The floor of the sella is eroded and sometimes destroyed completely. Though it is stated that the sella is normal in a suprasellar meningioma, because these characteristics are absent there must be changes in the clinoid processes or walls of the sella that are not now detected. The interested efforts of the roentgenologist are necessary in order that these differentiations may be made more easily.

In a series of 252 intracranial tumors, 191 of which have been verified by microscopic section, there have been 14 instances in which the lesions may be placed in the general group of chiasmal lesions. All of these patients were operated on. The operative mortality rate was 14.2 per cent, which means that 2 patients died in the hospital after operation. One of these deaths occurred in a case in which the patient had a glioma of the optic chiasm, and the other followed the removal of a craniopharyngioma in a boy, aged 10 years. Of the remaining 12 patients, 10 have lived for periods varying from ten years to nine months after operation. One child died four years following operation, and another patient with a glioma of the optic chiasm suddenly died three months after operation. Perhaps most important of all, 9 of the remaining 10 patients are socially and economically independent.

It is only by concentrated and concerted efforts that one may hope to improve the clinical results. Constant improvement in the surgical attacks on these lesions must go hand in hand with the earlier recognition of the pathologic changes by the roentgenologist and the ophthalmologist.

54 East Erie Street.

ABSTRACT OF DISCUSSION

DR. TEMPLE S. FAY, Philadelphia: I have had the misfortune to operate on a patient in whom the cerebellar signs, as well as the uniform ventricular dilatation, high choked disk and blindness, seemed to warrant the diagnosis of a lesion of the posterior fossa. The posterior fossa showed a large collection of fluid. At autopsy, eleven weeks later, this intense arachnoiditis above the chiasm was discovered. Seven other similar cases have been detected by means of the encephalogram after careful control of intracranial pressure and removal of ventricular accumulation of fluid. The adhesions in this area are produced by a low grade tuberculous basilar meningitis, as well as by inflammatory reactions secondary to hemorrhage. The question of sinus and mastoid infections and the leptomeningitis resulting from this is not to be overlooked. Four cases in our series were posttraumatic in origin, one tuberculous meningitis, and two others suspected of tuberculous meningitis but not verified. The confusing symptom-complex is often reduced to proper components by placing the patient on a limited fluid intake of a total of 20 ounces (591 Gm.) a day and dry diet, and observing the effect on the choked disk, intracranial pressure and focal symptoms. This has offered the best course of study in these cases and has made possible a prolonged investigation, which has aided in the establishment of a correct diagnosis. Such delay would not be justified in the presence of continued intracranial pressure. An active means of dehydration must be accomplished or visual loss becomes a matter of great concern. It has been possible to study cases presenting choked disk and intracranial pressure with actual recession in the choking in various tumor groups by fixing the fluid intake and placing the patient on proper dehydration. Dr. Davis's paper emphasizes the importance of careful visual field determinations and I feel that, with the addition of a fixed fluid intake and dry diet associated with the usual means of dehydration, a more constant and clearcut visual field picture will result, making interpretation and localization possible with greater precision.

DR. CLIFFORD B. WALKER, Los Angeles: Of the greatest interest to ophthalmologists and neurologists should be the observations recorded in case 7. The actual swelling and crowding of the optic nerve, so that it was cuffed over the optic foramen, were noted. The chiasm also was seen to be swollen as well as the optic nerve tissue itself in the case of acute onset with some, but not all, of the features of either retrobulbar neuritis or encephalitis. I reported a case of the latter sort some years ago and stated the probability of swelling of the optic nerve in its canal as the possible cause of blindness in case the canal was undersized. Roentgen measurements of the canals were used to support this argument. Dr. Davis, I believe, has added a further confirmative observation. I stated then that a decompression of the optic canal was the desirable maneuver if it could be done. I believe that Dr. Davis could have decompressed these canals and obtained still better results. That this can be done has been shown by Dandy and also this year by Dr. Howard Naffziger, who obtained a complete recovery of vision in both eyes after opening both the canals. In Naffziger's case, it is true that the cause was different (exophthalmos following thyroidectomy) but the fact remains that it can be done and really offers protection against blindness in certain instances. Since pathologic changes somewhat analogous to those of choked disk have been found in the membranous labyrinth with increased intracranial pressure, it is always worth noting the functional condition of the eighth nerve as to whether deafness, for instance, may become a chiasmal symptom in case of tumors in the region large enough to produce cerebral pressure. I would emphasize the value of x-rays in treating certain limited group of chiasmal lesions in poor surgical risks;

for example, a person of 70 years. The use of a range of disk sizes from 1/2,000 to 40/330, if necessary, to obtained 3 or 4 isopters after the manner of quantitative perimetry is important in order to follow more exactly the progress of the vision in these patients, most of whom value eyesight even more than life. Examination of the blind spot should be performed as a routine together with careful recording of the corrected vision on the chart as a record of the center of the field and an index of the observation power and fixation ability of the patient, as well as a step toward detection of scotomas. If the seeing field is recorded in solid black silhouette for one size only of test object, it is obvious that the question of status of most of the points mentioned which are most valuable in detecting changes in the visual progress of the patient are left without record.

Dr. T. B. Holloway, Philadelphia: I think that since Cushing's outstanding work in 1906 what has been found in regard to the pituitary body has been little short of a revelation. As the author has said, much has been brought about by improvement of surgical technic, but there has also been another factor and that is the concentration of trained specialists on this particular region. I was happy to note that the author was good enough to give the ophthalmologists and roentgenologists some credit for this work. Recently it has been my pleasure to go over the cases of adamantinoma occurring in the neurosurgical department of the university in the service of Dr. Frazier, and we have isolated about 14 of these tumors. Our chairman in his address referred to adamantinoma of the orbit. These adamantinomas comprise one of four types of tumor of the skull, the others being cranial tumors, carcinomas and keratomas. I want to show some types of fields found in these 14 cases of adamantinoma and then compare them briefly with the results found in 105 cases of adenoma of the pituitary which I had occasion to review several months ago. When we compare these fields we find optic atrophy present in 84 cases of adenoma and 48 cases of adamantinoma. Choked disk is present in 8.5 per cent of adenomas and in 51.4 per cent of adamantinomas. It is also interesting to note that in the cases of adamantinoma with choked disk the latter appeared before the age of 15 in 63.1 per cent of the cases.

Dr. Loyal Davis: I am deeply grateful to the men who have discussed this paper which, because of the shortness of time, was given in a great hurry. I want to take this occasion to point out that some of the progress in neurologic surgery may be attributed to the careful perimetric studies of Dr. Walker. The cases classified as arachnoiditis are also classed as suspected tumor because later they may show definite localizing symptoms of a tumor or at autopsy a tumor may be verified. With regard to the tumors that arise about the chiasm or from the third ventricle, these are the cases which the neurosurgeon looks on with great fear because frequently hyperthermia develops after an operative attack on the growth. Roentgen treatment of the pituitary adenomas is carried out in our clinic, but no pituitary adenoma is given this form of treatment without careful checking of the perimetric fields, because it has been shown that not infrequently after roentgen treatment of these tumor vision may disappear completely.

CHOROIDEREMIA

REPORT OF A CASE *

WALTER R. PARKER, M.D.
DETROIT
AND
F. BRUCE FRALICK, M.D.
ANN ARBOR, MICH.

Choroideremia may be classified as a bilateral developmental abnormality in which the entire choroid, except a small area in the region of the macula, is absent. The central vision may be normal, but there is always a marked contraction of the visual fields, usually associated with night-blindness. The condition was first described in 1872 by Ludwig Mauthner.[1] He disagreed with Leber, who thought that these cases could be classified in the retinitis pigmentosa group. While the pathology of these cases may not be definitely settled until more observations have been made, including a more thorough study of the histologic and functional characteristics of the condition, most observers believe that choroideremia is the result of a congenital defect and is not due to an inflammatory change.

A report of a case is presented, together with an unpublished case, notes of which are furnished by Dr. de Schweinitz, and a résumé of the salient points in the twelve cases previously reported.

REPORT OF A CASE

H. C. D., a man, aged 33, presented himself at the ophthalmic department of the University of Michigan Hospital on Oct. 24, 1930. His chief complaint was nyctalopia, which had been present as long as he could remember. He had always been nearsighted, and he stated that he had difficulty in locating objects unless they were straight in his line of vision. After the object was located he had no difficulty in seeing it clearly. Going from one intensity of illumination to another bothered him a great deal, and he was slow in adapting himself to slight changes in intensity of light. Bright lights dazzled him. He read without difficulty, and had no disturbance in central color vision.

The family ocular history was unimportant. There was no consanguinity. The patient had two sisters, neither of whom complained of visual disturbance. One sister was examined and was found to be normal. The past history of the

* Submitted for publication, March 20, 1931.

* Read before the Section on Ophthalmology at the Eighty-Second Annual Session of the American Medical Association, Philadelphia, June, 1931.

1. Mauthner, Ludwig: Ein Fall von Choroideremia, Ber. d. naturw.-med., Ver. in Innsbruck **11**:191, 1872.

patient was irrelevant, except that his eyes were refracted without cycloplegia in 1928, at which time vision was 10/200 in each eye; with a —4.00 sphere, vision was improved to 20/40 in each eye.

The external examination gave negative results. The irides were bluish green. Vision was 2/60 in each eye. The pupils were round and equal in size, and reacted normally. Transillumination gave one the impression of an albinic eye, due to the free transmission of light. In the right eye the near point was 12 cm. and in the left eye 11 cm. The amplitude of accommodation was 7.5 diopters in each eye. Muscular balance was normal. Tension was 18 in the right eye and —17 in the left eye (Schiötz).

Refraction under homatropine showed: in the right eye with a —3.50 sphere, cylinder —.75, axis 30, vision of 6/7.5, and in the left eye with a —3.00 sphere, cylinder —.75, axis 152, vision of 6/12. The visual fields were contracted to within the 3 degree circle in each eye. By means of Nagel's anomaloscope the patient was found to have moderate anomalous protonopic trichromic vision (moderate red-blindness in the lower degrees of intensity of red). For all practical purposes his color vision could be considered normal. An attempt was made by means of the Purkinje apparatus to determine the patient's power of adaptation to light. The results were somewhat unsatisfactory, however, as the field of vision was so constricted that it was not always possible to tell whether the patient no longer saw the light because of the low degree of illumination or whether his fixation had wandered. It seemed reasonably certain that the patient had some degree of adaptation to darkness.

Ophthalmoscopic Examination.—In the left eye, the pupil dilated evenly, the lens was clear, there were many fine floating opacities and the margins of the disk were blurred; the lamina cribrosa was not seen. The retinal vessels showed a normal distribution; the veins were full and the arteries contracted. The choroid was absent except in the macular region, giving the whole fundus a pearly-white appearance outside the central area; the latter showed a plexus of choroidal vessels surrounded by a fine pigmented region that gradually faded into the white background, except temporally where the slightly pigmented area extended into the periphery. The central plexus of vessels had its origin in a perforating vessel that appeared immediately temporally to the border of the disk and divided into two branches which ran up and down for a short distance parallel to the margin of the disk, then turned abruptly toward the macula. In the macular region these two arteries divided and formed a vortex-like arrangement from which two branches continued in a more or less horizontal direction toward the temporal periphery. From the point of entrance and the course taken by this vessel, it apparently was a short posterior ciliary artery. Another vessel perforated the sclera immediately above the macula and ran up and out, nearly parallel to a descending branch of the superior temporal artery of the retina. About 2 disk diameters mesially from the border of the disk there was a small spot of pigment over the retinal vessels. In the extreme temporal periphery a large plexus of vessels was seen from which two branches emerged which diminished in size as they coursed toward the macula and became lost in the central area. They appeared to come from the ciliary body, and apparently were branches of the long anterior ciliary artery. No long posterior ciliary arteries or vorticose veins were seen. Several small accumulations of pigment were scattered irregularly about the fundus. The general appearance of the right eye was the same as that of the left. The arrangement of the vessels carrying blood to the macular region was almost identical.

Reproduction of drawing of the fundus in the case of choroideremia reported.

Dr. de Schweinitz supplied the notes on the following case of choroideremia:

J. B., a boy, aged 14, was referred for examination by Dr. Homer J. Rhode, who reported that the patient had always had poor vision, and that the condition was considered as congenital. The parents were both healthy and, as far as known, were not related to each other.

The first examination was made on Feb. 4, 1921, at which time the patient was 14 years old. There was a constant lateral nystagmus in each eye. There were some anterior cortical opacities in each eye associated with a somewhat star-shaped posterior cortical opacity. The ophthalmoscope revealed practically complete choroideremia except in the area surrounding the macula. The condition was more marked in the right than in the left eye.

In the right eye refraction with a —7.50 sphere, cylinder —1.50, axis 180 showed a vision of 5/60; in the left eye, with a —7.50 sphere, cylinder —1.50, axis 180, a vision of 5/50. The patient could read M at 2 feet (60 cm.) with difficulty. There was marked night-blindness.

The patient was reexamined in March, 1921, February, 1922, and October, 1927. At the last named date there had been a considerable increase in the cataractous process, which had now changed to large white plaques surrounded by numerous spokelike opacities. Vision was materially reduced, being 20/60 in the right eye and 1/60 in the left. Cataract extraction was advised and successfully performed by Dr. Rhode, the resulting vision being 6/30 in the right eye and 2/60 in the left. The patient could read M at 1 foot (30 cm.).

The field of vision taken in 1928 with a 20 mm. white object at 300 mm. was contracted to within the 20 degree circle. The patient is now 24 years of age, and as far as known, his vision remains the same. He is able to carry on his regular work.

Twelve cases have been reported since Mauthner's original communication (see table). These have been reviewed by Nettleship [2] and Wolf,[3] and the details of these records will not be repeated. It is interesting, however, to note the uniformity in the observations recorded. With but one exception the patients have all been males between the ages of 16 and 44. They give a definite history of bilateral, unchanging, defective sight since childhood, not antedated by illness or injury to which it might be ascribed. No history of consanguinity was mentioned, and in six of the twelve records it was specifically stated that the parents were not related. One brother of Mauthner's patient was said to have a similar defect of vision, but was not examined. Koenig [4] and Wolf each reported the condition in two brothers. Thompson's [5] patient had a great uncle who suffered from night-blindness. No succes-

2. Nettleship: On Retinitis Pigmentosa and Allied Diseases, Roy. Lond. Ophth. Hosp. Rep. **17**:373, 1907-1908.

3. Wolf, S.: Choroideremia, Arch. Ophth. **3**:80 (Jan.) 1930.

4. Koenig: Zwei Beobachtungen von mangelhafter Entwicklung der Choroides verbunden mit Hemeralopia, Inaug. Dissert., p. 17.

5. Thompson: Congenital Deficiency of the Choroid, Tr. Ophth. Soc. U. Kingdom **19**:140, 1898-1899.

Data on Cases of Choroideremia

Author	Date	Sex	Age	Symptoms	Vision	Refraction	Fields	Color Sense	Light Adaptation	Heredity	Consanguinity	Comment
Mauthner	1872	M	32	Nearsightedness; nyctalopia	5/200 10/ 40	—3.00 —2.00	Contracted within 20 degrees	?	Poor	?	?	Glistening white fundus except at macula; some pigment
Koenig	1874	M M	20 ?	Nyctalopia Nyctalopia	?	?	Contracted to 40 to 70 degrees	Good	Poor	One brother with same condition	None	Absence of choroid except at macula; some pigment
Cowgill	1892	M	17	Poor vision always	10/200 8/200 with —8.00	—13.00 —16.00	?	?	?	?	?	Scleral whiteness except at macula
Bullar	1898	M	16	Poor vision always	6/18 6/12	—5.50 —5.50	Contracted to 10 degrees; peripheral part 10 degrees wide	?	?	?	None	Grayish-white fundus with scattered pigment dots; red macula
Thompson	1898	M	18	Myopia; tubular vision; nyctalopia	6/9 6/9	—3.00 —3.00	Contracted to less than 10 degrees in both eyes	?	Poor	Great uncle had nyctalopia	None	White fundus except at macula; some pigment
Landman	1905	M	44	Poor vision always; nyctalopia	6/8 6/18	—1.00×90 Not improved	Contracted to 3 degrees	Good	Poor	?	?	Shiny white fundus except at macula; some pigment
Wardale (Northumberland & Durham M. J. Newcastle-upon-Tyne **14**: 3, 1906)	1906	M	26	.....	C.F. 4/60	—7.00	.....	.....	.....	.....	.....	Case reported by Thompson in 1898; appearance of fundus the same but vision —7.00 right eye; C.F. 4/60, left eye
Marbaix (J. méd. de Brux. **13**: 475, 1908)	1906	M	40	Nyctalopia	1/3	—3.00 —4.00 × 90 —3.00	Contracted from 80 to 20 degrees	Good	Poor	None	?	Scleral white fundus except at macula
Grimsdale	1916	F	23	?	6/60 6/60	—5.5 —3.00	Greatly contracted	?	?	?	?	White fundus except around disk and macula; pigment patches flat
Connor (Am. J. Ophth. **2**: 553, 1919)	1919	M	25	Nyctalopia	20/20 20/20	+0.50	Markedly contracted, from 60 to 30 degrees	Good	Poor	None	None	Glistening white fundus except at macula; pigment dots
Wolf	1930	M	40	Poor vision always	20/100 20/100 20/20	—1.75 (both eyes) —1 sph. —1 cyl. × 90	Contracted to less than 10 degrees	Good	Poor	None	None	Greenish-white and red macula; some pigment
		M	42	.....	20/30	—1.5 cyl. × 90						
de Schweinitz	....	M	14	Myopia; nystagmus; nyctalopia	5/60 5/50	.....	Contracted within 20 degree circle	?	?	None	None	Cataract in each eye; operation on right eye; final vision, 6/30
Parker and Fralick	1931	M	33	Nearsightedness; limited vision; nyctalopia	6/7.5 6/12	—3.50 —.75 × 30 —3.00 —.75 × 152	Contracted to 3 degrees	Good	Poor	None	None	White fundus except at macula; scattered pigment

C. F., counts fingers.

sive generations have been followed, however, to establish an hereditary basis for the anomaly. The presence of night-blindness and lack of adaptation to light was mentioned by all others except Cowgill,[6] Bullar [7] and Grimsdale.[8] Cowgill, alone, did not mention the contraction of the form fields with the preservation of good perception of color. All the authors reported the absence of the choroid except in the macular region. While the retinal vessels were not involved, isolated choroidal vessels were sometimes seen in parts of the fundus other than the macular region (probably the long posterior ciliary artery), and vorticose veins were described. Scattered pigment in various parts of the fundus was noted in all the cases. In one case (de Schweinitz') cataracts developed and an operation was performed successfully.

COMMENT

A histologic study of our case could not be made, and it is therefore impossible to add anything to the various theories that have been advanced for the occurrence of choroideremia. It is interesting to contrast the appearance of the eye in this condition with that in coloboma of the macular region. In what is termed macular coloboma there is a round or oval patch at the macula, the remainder of the fundus presenting the normal red reflex, while in choroideremia the reverse condition is present; namely, the central area remains normal in appearance while all the rest of the choroid is absent. This appears to confirm the possibility of the macula having an independent vascular system, as suggested by Marbaix. But as a matter of fact, colobomas may be present in any portion of the choroid, the appearance of the area involved being similar to that seen in choroideremia, though less extensive. Landman [9] suggested that the macula plexus was probably supplied by branches of the posterior ciliary arteries which passed directly through the sclera and did not come out at the edge of the disk like the usual cilioretinal vessels.

There evidently is a faulty development of the vascular system of the choroid, but just how or why this occurs is not known. Collins and

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6. Cowgill: Report of a Case in Which There is Binocular Absence of the Choroid, Showing a Rich Plexus of Cilio-Retinal Vessels Supplying the Region of the Yellow Spot in Both Eyes, Arch. Ophth. **21**:105, 1892.

7. Bullar: A Case of Deficiency of the Choroid, Tr. Ophth. Soc. U. Kingdom **18**:183, 1897-1898. (The particulars were furnished by Bullar to Nettleship and were also reported by him.)

8. Grimsdale: Unusual Condition of the Choroid (? Congenital), Proc. Roy. Soc. Ophth. **10**:29, 1916.

9. Landman: A Case of Symmetrical Congenital Absence of the Choroid and Retina Except in the Macular Region, Arch. Ophth. **34**:473, 1905.

Mayow [10] stated that a failure in the development of the choroid is due to a failure in the formation of blood vessels in the inner part of the mesoblast which surrounds the secondary optic vesicle. The choroid receives its chief supply of blood from the short ciliary arteries. A failure in the formation of all these vessels, except those that supply the region of the macula, would account for the condition described as choroideremia. The large ciliary vessels, which are sometimes seen in cases of coloboma of the choroid coursing over the affected area, are most likely branches of the long posterior ciliary arteries. Some of them may also be veins proceeding from the ciliary body to the vortex veins which perforate the sclerotic, just posterior to the equator. According to Mann,[11] the formation of the choroidal net appears with the pigment and in some way is apparently related to it, as shown by the fact that, if pigment is absent over an area of the surface of the optic cup for any reason, the choroid also is absent. "It seems best that typical coloboma of the choroid be considered as due to an arrest or delay of development of the lower part of the optic cup, associated with aberrant growth of the margins of the foetal fissure. Colobomata may occur in other regions of the eye, but very little is known to account for this. It is better not to consider such atypical patches of retinal and choroidal atrophy as having any connection with the cleft or with normal development at all. A certain number of them may be explained as a result of intra-uterine disease, while others are possibly due to hemorrhage at birth." [12]

CONCLUSION

About all that can be said at present, then, is that coloboma of the choroid may be attributed to the absence of the short ciliary arteries supplying the particular region affected, and that the condition is developmental rather than pathologic; also that the clinical appearance of the fundus in choroideremia and in coloboma of the macula suggests the possibility of an independent vascular supply for the macular region.

ABSTRACT OF DISCUSSION

DR. EDWARD B. SPAETH, Philadelphia: If inflammatory factors may be disregarded in the etiology, choroideremia should be considered congenital and due to a developmental defect. In the choroidal defects seen in choroideremia one must of necessity conclude that the development of the associated mesoderm plays an important rôle. I believe Parker to be wholly correct in stating that colobomas of the choroid must be in some manner connected with the faulty development of the blood vessel anlagen which lie in the fetal cleft.

10. Collins and Mayow: Pathology and Bacteriology of the Eye, Philadelphia, P. Blakiston's Son & Company, 1911.

11. Mann, Ida C.: The Development of the Human Eye, New York, The Macmillan Company, 1928, p. 39.

12. Mann (footnote 11, p. 147).

Connor, in reviewing the etiology and embryology of choroideremia, stated: "The analog in embryonic life of what is later the macular area of the fundus is a spot on the outer surface of the secondary optic vesicle where the first signs of differentiation of the mesoblast into sclera and choroid are seen, the first appearance of the choroid being manifest by a tiny plexus of capillaries at this point." Hence, in seeking an explanation of the cause of choroideremia, we must consider that differentiation of the mesoblast of the secondary optic vesicles began in a normal way and at the normal point, but for some reason was arrested when only the macular area was supplied with choroid.

There is still a third possibility to be considered. The inconstancy of anatomic structures is well known. This applies frequently to the vascular system of the body, in both the gross and the microscopic. Further, as Leber stated, there is a close relationship between the three blood vessel systems of the eye: the first, that of the choroid, the ciliary body, and the iris; the second, the blood supply of the retina and the optic nerve, and the third, that of the conjunctiva and the orbit. Further details of this are unnecessary here. We have all seen this close interrelationship in the congenital arterial anastomoses described by Leber, by Schleich and by Seydel, in the peripapillary vascular ring described by Coppez, in the central retinal artery anomalies described by Ginsberg and Terrien and by Hirsch, and in the many remnants of the hyaloid artery and the tunica vasculosa lentis described by many authors. Such irregular and inconstant vascular defects might also be a factor in the etiology.

Dr. Harry M. Weed, Buffalo: A recent canvass of many of the principal blind institutions throughout this country fails to show that any patient with congenital absence of the choroid has ever been diagnosed in any of them. Thus far, little more has been done on this subject than to recognize fourteen cases in nearly sixty years, all bilateral, all but one myopic, and only one, the case of Dr. de Schweinitz, associated with any other congenital defect. The authors have well pointed out the essential difference between congenital absence of the choroid and those developmental defects related to faulty closure of the cleft in the optic vesicle, calling attention to the fact that in the one it is the macular region only that functions in vision, while in the other the macula is most likely to be affected with absence of central vision.

Discussion as to the distribution and growth of mesoblastic fetal tissue about the optic vesicle is futile, and equally so is the fact that pigment does not appear in the choroid until late in fetal life. It would seem strange that direct ancestral history has been obtained in none of these cases, and perhaps equally strange that no offspring with similar defects has been discovered, as most of the patients in the reported cases were of marriageable age when first observed.

This puzzling condition will be understood to some degree only when some alert ophthalmologist succeeds in obtaining one of these eyes for histologic study. In the meantime, each case that is discovered should be thoroughly investigated as to ancestry and followed carefully as to progeny. It has been proved that the law of Mendel applies to all other congenital ocular defects, and it is inconceivable that the same law should not apply to congenital absence of the choroid.

Dr. Arthur J. Bedell, Albany, N. Y.: Choroideremia is of special interest to me for several reasons: first, because of my good fortune in being permitted to examine and photograph Dr. Parker's patient; second, because of the rarity of the condition as reported in the literature; third, because as far as we know, in only three instances have the eyes been photographed, or at least the cases published; and lastly, because I believe choroideremia can appear in more than one form.

The condition as recognized in the photographs of Dr. Parker's patient is an absence of a zone of the choroid, a degeneration of a wide peripheral circle and the retention of a small ovoid central area of choroid. It would seem that this central area remained active because of an evident independent vascular supply of the macular region. The photographs clearly portray the vessel which Dr. Parker has commented on to be separate and distinct from the retinal circulation, and we can by means of the photograph see that the circulation of this central macular area is supplied by an artery that comes from below, and is drained by a vein that enters above the entrance of the artery. This in itself is of sufficient interest to warrant the attention that has been attracted to this condition. Especially is it true in the present case because, as can be recognized, the same vascular anomaly is present in each eye.

We must seriously consider Leber's primary suggestion that some of the cases of choroideremia can be classified in the retinitis pigmentosa group.

There is at least one other condition that may probably be called choroideremia. This is definitely an acquired disease and consists of an absorption of the choroid. One such case we illustrate with the photograph of a patient of 63 who had syphilis for several years. It will be noted in the picture that the choroid is practically entirely absorbed, that the retinal and choroidal pigment has migrated to form isolated, more or less rounded masses and that the arteries and veins are attenuated and the disk muddy gray. This form can be easily differentiated from the type described by Dr. Parker by the fact that it is unilateral, that it seems to be an acquired lesion and that the night blindness is not as extreme, but that all parts of the choroid are involved. There can be no confusion between choroideremia and coloboma of the choroid because in coloboma of the choroid the edge is always sharply delimited, and the colobomatous zone depressed.

Dr. Edward Jackson, Denver: The vascular system in general has great powers of recovery, and the development of the vessels of the eye, as has been so admirably set forth by Miss Ida Mann, is intimately associated with the stimulation of the epiblastic structures of the eye — the retinal development. The development of the macula occurs at a later period, embryologically, than the vessels of the interior of the eye or the choroid. The macula, as Miss Mann showed, is not developed at birth; the child is 4 or 5 months old before macular development is complete. Now it is possible — and this should be borne in mind with reference to infection — that a temporary condition affecting the mother might prevent the development of the general choroidal circulation, but subsequent recovery might allow macular development.

One other point is the matter of night blindness. Night vision is associated with rods; the macula is a cone region. It is entirely possible that night blindness is merely an absence from all parts of the eye of any considerable area of rods, and on that account the vision by feeble or artificial illumination is very inferior.

SOME CONTRIBUTIONS TO THE ART OF LOCALIZING OCULAR FOREIGN BODIES*

HARRY A. GOALWIN, M.D.
NEW YORK

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MERIDIONAL LOCALIZATION: LOCALIZATION WITH RESPECT TO STRUCTURES OF EYE

When an ocular foreign body is localized from roentgenograms by one of the graphic or mathematical methods, its position is described by giving the horizontal and vertical components of its distance from the optical axis and its horizontal distance from the apex of the cornea.

Translated into mathematical terms, the foreign body is localized with reference to a system of rectangular coordinates, the origin of which is at the apex of the cornea. In this system the X axis is the horizontal tangent to the apex of the cornea, the Z axis is the vertical tangent to the apex of the cornea, and the Y axis is the normal to the apex of the cornea (which coincides with the optical axis). The horizontal meridian section of the eyeball is therefore in the x-y plane, the vertical meridian section is in the y-z plane, and the equatorial section is parallel to the x-z plane.

It is customary to indicate the position of the foreign body on three views of the eyeball. Each of these views, being parallel to one of the coordinate planes, is a projection of the full eyeball on that plane. The front view is therefore equal in shape and size to the equatorial section, and the side and top views are equal in shape and size to the

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meridional section. It is quite evident from the laws of projection that on a sketch of this kind a foreign body may appear within the outline of the eyeball on all three views and yet be actually extra-ocular.

Take, for example, a point 15 mm. from the center of the eyeball, hence extra-ocular, situated so that the radius prolonged passing through it is equally inclined to all the principal axes. The three projections of this 15 mm. line will be of the same magnitude and equal to 8.65 mm. (the square root of ⅓ of 15^2). The foreign body located at this point will therefore appear to be within the eyeball in all three views.

This erroneous representation can be avoided if the location of the foreign body is indicated on sectional views that pass through it. Such a method has indeed been proposed and used by Stumpf,[1] who employs a chart of transparent cross-section paper on which has been printed a series of sagittal sections of the eyeball taken 1 mm. apart. Each of these sections naturally differs from the others in size and shape. To avoid confusion, the final report would have to contain only the section that passes through the foreign body. This would necessitate the preparation of a separate drawing for each localization —a method that is possible and practical, but by no means economical. I have combined the advantage of the precision of this method with the economy and simplicity of the Sweet chart (modified by Dixon) in the method of meridional localization.

The theory on which this method is based is extremely simple: A meridional section of the eyeball is constant in shape and size for a given individual, irrespective of the direction of the meridian. If one takes the meridional section which passes through the foreign body and indicates the location of the foreign body in its plane, then it can be seen at a glance not only whether the foreign body is intra-ocular or extra-ocular, but also what particular structure of the eyeball it is in (if intra-ocular). The further advantage of this method is that it involves no immediate abandonment of the almost universally adopted method of localization and plotting of ocular foreign bodies. The roentgenograms are made and the foreign body is localized in the usual manner (Mackenzie-Davidson, Sweet, Dixon). The foreign body is then indicated on the present chart in the usual manner.

On the anterior view a line is then drawn which passes through the center of the eyeball and through the foreign body. This line indicates the direction of the meridional section concerned. On a sketch of the meridional section a perpendicular is then drawn to the axis at the same distance from the apex of the cornea as was done in the top and side views. From the foot of this perpendicular (its intersection with the axis) as a center, and with a radius equal to the distance

1. Stumpf: München. med. Wchnschr., 1916.

from the center of the eyeball to the foreign body, as measured on the anterior view, an arc is drawn which crosses the perpendicular above the axis if the foreign body is above the horizontal principal plane, or below the axis if the foreign body is below the horizontal principal plane. The intersection of the arc with the perpendicular indicates the exact site of the foreign body and its relation not only to the outer walls of the eyeball, but also to the inner structures (ciliary body, lens, etc.).

AN IMPROVED CHART

I have designed a new chart on which one can combine the meridional localization with the established method. Fundamentally, this chart looks like the present charts, except that it contains a meridional section in each upper corner. There are certain essential differences, however, some of which are not apparent in the finished product:

1. The dimensions employed are not those of the traditional schematic eye (of Helmholtz), but those of the more precise schematic eye of Gullstrand.

2. The meridional sections show the equivalent nuclear lens of Gullstrand, the iris and ciliary body, and thickness of the ocular wall.

3. The chart is prepared by reduction from a large chart which has been drawn to a linear scale of 10:1. The latter increases precision by reducing the possible error one-tenth; for it is obvious that a difference of, say, 0.25 mm. in the length of a natural size drawing of an eye represents an error of about 1 per cent, while the same difference of 0.25 mm. in a drawing made to a scale of 10:1 would represent an error of 0.1 per cent.

4. The anterior views are surrounded by a scale of angles, indicating the directions of the meridians 10 degrees apart, with an arrow at each 30 degrees.

5. On the reverse side of this chart is another chart, showing the anterior view of the eyeball and the meridional section enlarged two and one-half times.

THE SQUARE ROOT RULE

I have already stated that a foreign body may appear to be within the eyeball in all three views of the present charts and yet be extra-ocular. There are occasions when one may wish to determine whether a foreign body is intra-ocular without resorting to meridional localization: 1. The roentgenologist himself may wish to check his observations by another method. 2. A meridional section chart, or indeed any other type of chart, may not be at hand. 3. The ophthalmologist may wish to check the localization. 4. The ophthalmologist may receive

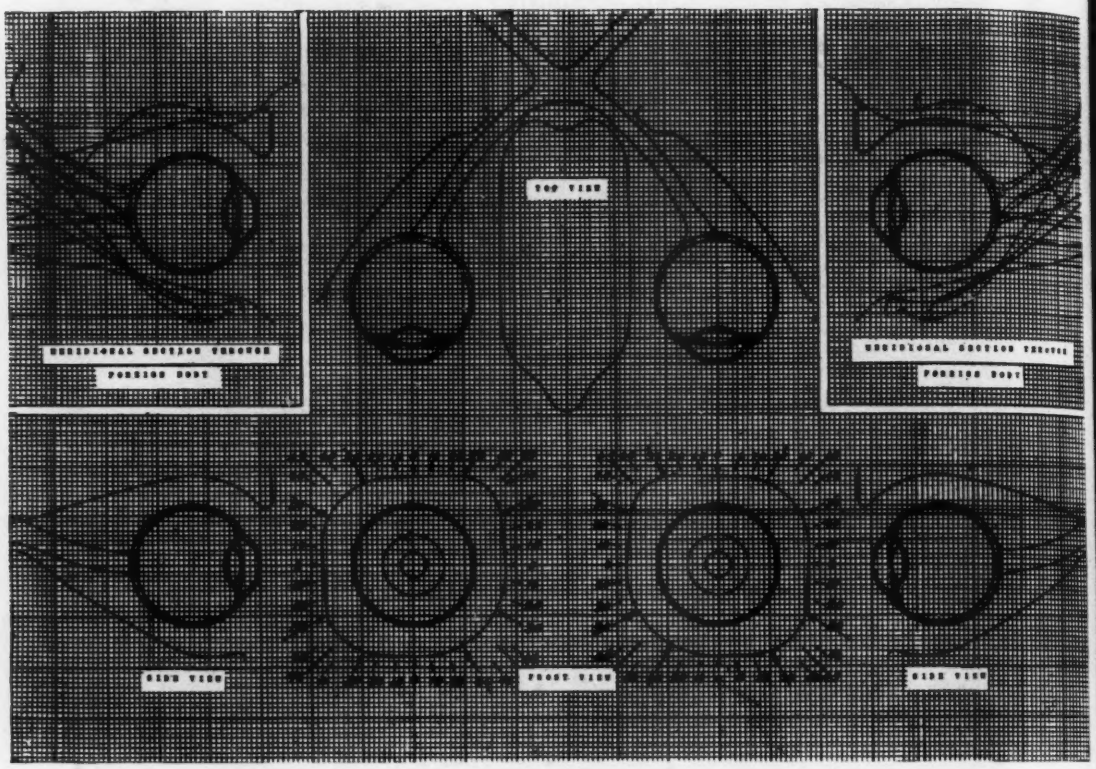

Fig. 1.—Localization chart with the meridional section in each upper corner. The actual size is 6 by 9 inches, and occupies the lower half of the 9 by 12 inch sheet, the upper half being reserved for the letter head and written report. Scale, 1 : 1 (in actual practice).

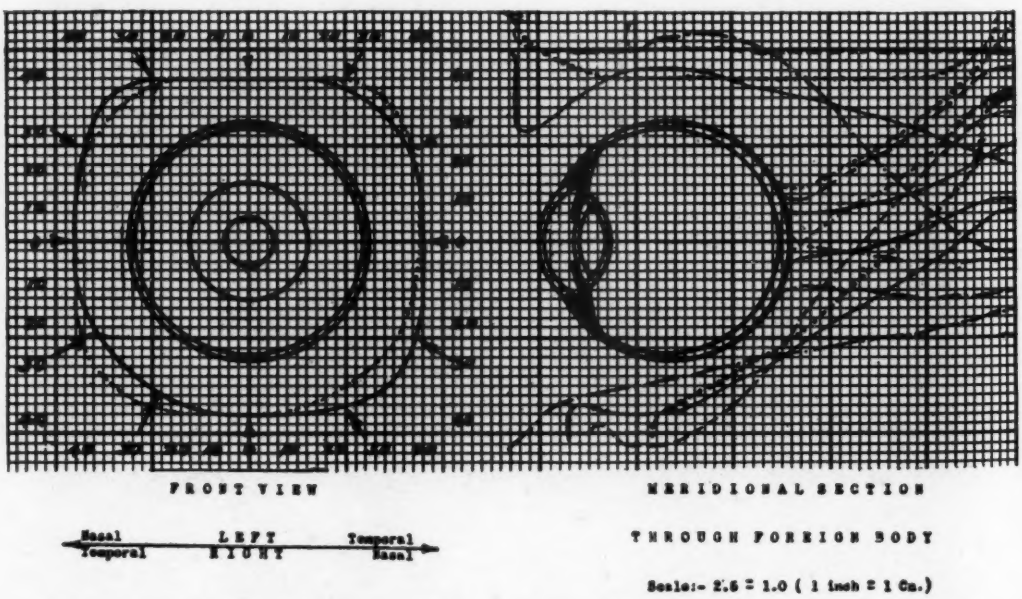

Fig. 2.—Meridional localization chart which is on the entire reverse side of 9 by 12 inch sheet described in figure 1. The eye and orbit are enlarged two and a half times.

(as he undoubtedly will for a long time to come) a report with the conventional chart, without a meridional localization. For this purpose I use the square root rule.

The theory of this rule is as follows: The radius of the retinal surface of Gullstrand's schematic eye is 11.4 mm., and the center of this surface is 13 mm. from the apex of the cornea. The origin of my system of coordinates being at the apex of the cornea, it follows that the equation of this spherical surface is

$$X^2 + Z^2 + (13 - Y)^2 = (11.4)^2$$

or

$$\sqrt{X^2 + Z^2 + (13 - Y)^2} = 11.4$$

If 1 mm. is allowed for the thickness of the ocular wall, the following equation is obtained:

$$\sqrt{X^2 + Z^2 + (13 - Y)^2} = 12.4 \text{ mm.}$$

From this equation, the square root rule is derived as follows:

Let a foreign body be located *a* millimeters temporally or nasally from the vertical meridian plane, *b* millimeters above or below the horizontal meridian plane and *c* millimeters behind the apex of the corneas. Subtract *c* from 13 and add the square of the difference to the squares of *a* and *b*, then extract the square root of the sum. If the result is 12.4 mm. or less, the foreign body is intra-ocular; if more than 12.4, it is extra-ocular. If the result is between 11.4 and 12.4 mm., then the foreign body is in the ocular wall, the difference between the result (which is really the distance of the foreign body from the center of the eyeball) and 11.4 being the distance of the foreign body from the retina. If the distance of the foreign body from the center of the eyeball is more than 12.4 mm., subtract 12.4 mm. from it to obtain the distance of the (extra-ocular) foreign body from the eyeball. It must be remembered that the thickness of the ocular wall decreases gradually as one proceeds from the posterior pole forward, particularly in front of the equator. However, that will be discussed under the title of "Borderline Localizations."

This rule applies when the distance (*c*) of the foreign body from the apex of the cornea is 3.6 mm. or more. When *c* is less than 3.6 mm., one is in front of the plane of the limbus. Here a foreign body is bound to be either on the cornea or in the eyeball, unless its distance from the axis is 6 mm. (the radius of the limbus circle) or more. To obtain this distance, the squares of *a* and *b* are added and the square root extracted. If the result is more than 6 mm., the foreign body is in one of the lids or in the conjunctiva.

A foreign body may be in this prelimbus segment of the eyeball and yet be hidden from view, particularly in the presence of hemorrhage,

exudate, forward dislocation of the lens or other consequences of trauma. If one wishes to determine the perpendicular distance between the foreign body and the anterior surface of the cornea, the procedure is as follows:

The radius of the anterior surface of Gullstrand's schematic eye is 7.7 mm. Its equation, the origin of coordinates being at the apex, is

$$X^2 + Z^2 + (Y - 7.7)^2 = (7.7)^2$$

Since Y is always less than 3.6, hence less than 7.7, Y-7.7 will be a negative quantity. However, the square of a negative quantity is always positive, hence one may write

$$X^2 + Z^2 + (7.7 - Y)^2 = (7.7)^2$$

or

$$\sqrt{X^2 + Z^2 (7.7 - Y)^2} = 7.7$$

The square root rule for this segment therefore is obtained in the following manner:

Subtract c from 7.7, add the square of the difference to the squares of a and b, and extract the square root of the sum. The difference between this result and 7.7 is the distance of the foreign body from the surface of the cornea, measured along the corneal radii. If this difference is positive, then the foreign body is extra-ocular, hence in one of the lids or in the conjunctiva. If the difference is negative, then the foreign body is in the eyeball. To avoid the algebraic notation, this rule can be rephrased as follows:

If the square root of the sum of the squares is more than 7.7 mm., the foreign body is extra-ocular; subtract 7.7 mm. from this square root to obtain the distance of the foreign body from the cornea. This is measured along the prolongation of the radius which passes through the foreign body. If the square root of the sum of the squares is less than 7.7 mm., then the foreign body is intra-ocular; subtract this square root from 7.7 to obtain the radial distance of the foreign body from the anterior surface of the cornea.

CALCULATING THE SIZE OF A FOREIGN BODY

When the two ends of a foreign body have been localized the actual length of that body can be calculated. Let a, b, c and a', b', c' be the coordinates of the ends, then, if l is the length of the foreign body,

$$l = \sqrt{(a\text{-}a')^2 + (b\text{-}b')^2 + (c\text{-}c')^2}$$

So far I have discussed the localization of foreign bodies on two assumptions: (1) that my method is one of perfect precision, and (2) that I am dealing with an emmetropic eye. It is obvious that both of these assumptions are unwarranted by the actual conditions. I shall

disregard the first of these assumptions. According to Dixon (verbal communication), localizations are correct to within 2 mm. This means that one may err 1 mm. each way. I believe the error is less than that, only 0.5 mm, each way. Take a case in which the localization shows a foreign body to be 12.5 mm. from the center of the eyeball. Its actual position, even with the highest possible precision, is between 12 and 13 mm. from the center of the eyeball, hence it may be just within or just without the eyeball. Such cases are termed borderline localizations.

BORDERLINE LOCALIZATIONS

There are certain simple tests that one can apply, from which a fairly accurate conclusion can be drawn as to the actual location of the foreign body in a borderline case.

Most foreign bodies are not globular. They are of sufficiently irregular outline to present at least two sharp points for localization. When each end of the foreign body has been localized, one can calculate the distance of each from the center of the eyeball. If these two radial distances are equal or nearly equal, the axis of the foreign body is parallel to the surface of the retina; one may conclude that the foreign body is intra-ocular. In such cases the foreign body is usually in the lower half of the eyeball. If the two radial distances are unequal, the difference between the two being nearly or actually that of the length of the foreign body, it is an indication that the foreign body is perpendicular to the retinal surface. Since there is normally nothing in the interior of the eyeball (behind the ciliary body) to support a foreign body in such a direction one may conclude that it is within the ocular wall, at least partly.

When a foreign body has perforated the eyeball and passed on to the retrobulbar space, actually freeing itself from the globe, it will become inclined toward the ocular wall, due to the continual movements of the retrobulbar tissue.

The foregoing principles, together with an understanding of mechanics, of the nature of the process that was responsible for the entry of the foreign body into the eyeball, and similar considerations, will help make the borderline localization more definite.

If the foreign body is globular, so that one can localize its center only, then the procedure is as follows:

A profile roentgenogram of the orbit is made with the eye directed straight forward. Then the eye is directed toward a point at a measured distance downward and forward, so that one can reconstruct the angle, and take another profile roentgenogram. The foreign body will move, whether it is extra-ocular or intra-ocular. The lead indicator shows the location of the axis of the globe in the first roentgenogram. The

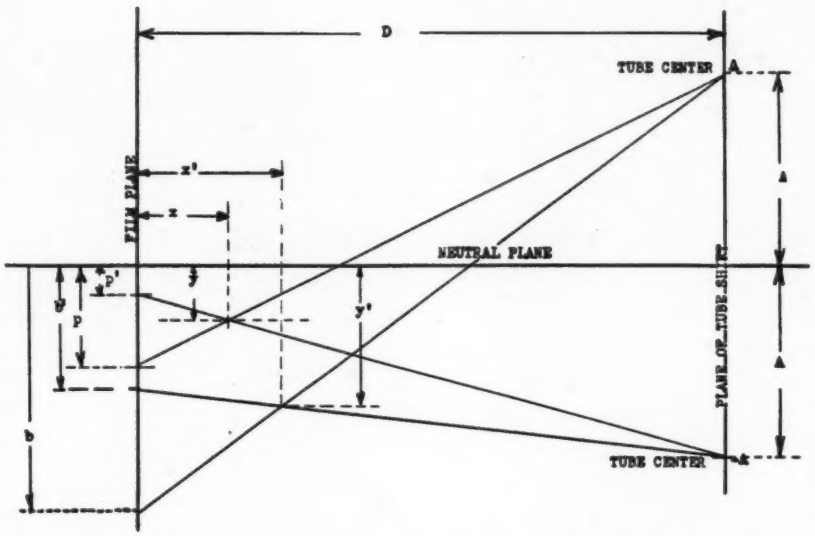

Fig. 3.—Sketch showing graphic determination of the anteroposterior and lateral positions of a foreign body. The mathematical formulas for localization are derived from this and the following sketch (not to scale).

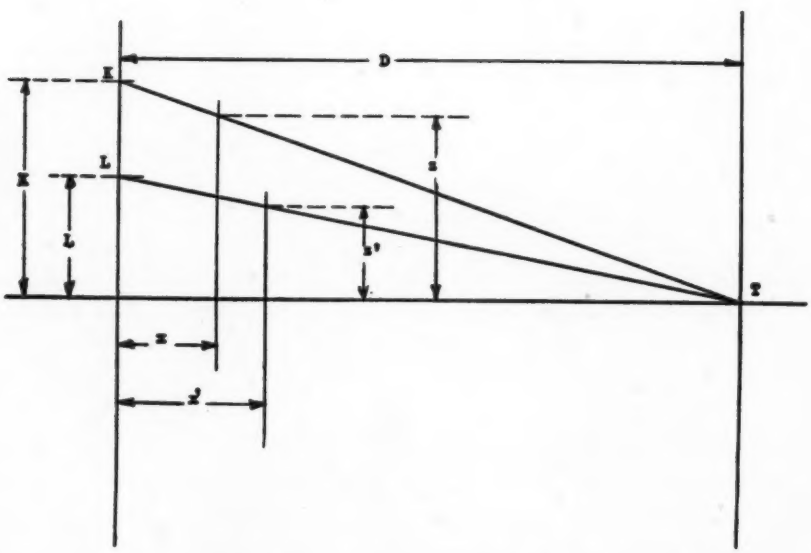

Fig. 4.—Sketch showing graphic determination of the vertical position of a foreign body. The mathematical formulas for localization are derived from this and the preceding sketch (not to scale).

locations of the two shadows of the foreign body are transferred to a sheet of paper on which the point of the indicator has been drawn. If a perpendicular bisector to the line connecting the two shadows of the foreign body is now drawn, then its intersection with the axis of the eyeball will indicate the center of the globe. If the two pictures have been taken with the meridional section passing through the foreign body parallel to the plate, then one can measure the distance of the foreign body from the ocular center and see whether it is intra-ocular or extra-ocular. When the meridional section just mentioned remains parallel to the plate, then the angle subtended by the two radii from the ocular center to the two shadows of the foreign body is equal to the deviation between the optic axes. If the former angle is less than the latter, then the meridional section was inclined to the plate and the ratio between the two angles will give the degree of this inclination.

This method naturally involves an additional roentgen examination, which is not always possible or practicable. One should therefore exhaust all other possibilities before resorting to it. A logical step in this direction is to increase the precision. I have devised for this purpose the method of two scale plotting.

TWO SCALE PLOTTING

To understand this method, one must remember the following facts. When making the roentgenograms of the orbit for localization of the foreign body, the center of the x-ray tube is about 50 cm. from the plate (47.5) and the indicator, which is over the center of the cornea at a known distance from it (from 2 to 10 mm.), is about 5 cm. from the plate. The tube is shifted about 6 cm. between the two exposures. The foreign body will be within 2 cm. on either side of the point of the indicator. Each will produce two shadows (one on each picture). A straight line is drawn from each shadow to the point which represents the position of the center of the tube when it produced that shadow. The intersection of the two lines from the shadows of the indicator point indicates the lateral position of the indicator point, hence also that of the apex of the cornea. The lateral position of the foreign body is determined in a similar manner. Now, since the planes in which these intersections will occur are not likely to be any nearer than 40 cm. to the center of the tube, the angles one has to deal with will be less than 10 degrees. When two lines intersect at so small an angle, the observer must have a very keen eye to judge within 0.25 mm. just where they do intersect, even when the lines have been drawn with a point of razor sharpness. One's precision can be increased in direct proportion to the increase in the angles of intersection. In order to accomplish this end, I leave the horizontal measurements undisturbed, so that distances along the horizontal axis are to a scale of 1 : 1, but enlarge the vertical distances

to a double or triple scale. In this manner the angles of intersection are in the vicinity of 20 or 30 degrees, and one can tell without difficulty just where two lines intersect, to less than 0.25 mm. It is obvious from the principles of geometry that the distances of the points of intersection from the plane of the plate will remain the same, irrespective of the vertical scale. The same applies to the distances from the plane in which the center of the tube is shifted. However, when measuring vertical distances one must remember the scale used, and make the appropriate reduction.

MATHEMATICAL LOCALIZATION

The graphic method is not the only one available for the localization of ocular foreign bodies. The mathematical method will give equal or even greater precision, provided the distances involved are measured with proper accuracy. Besides, there may be occasions when the drafting method cannot be resorted to.

Localizing roentgenograms have two straight lines which have been produced by the two wires that have been interposed between the plate and the skull. One of these is vertical and represents the plane along which the center of the tube has been shifted. The other is horizontal, and represents the horizontal plane midway between the two tube levels, which I shall call the neutral plane.

Let p and p' represent the distance of the lower and upper shadows of the indicator point from this neutral plane, and A the distance of the tube center from the neutral plane, so that the distance between the positions of the centers of tube is $2A$. Distances above the neutral plane are positive, while distances below this plane are negative. The upper tube position is therefore at plus A, and the lower tube position is at minus A; p and p' are positive or negative, depending on the relation to the neutral plane. They are usually below this plane, hence negative.

I shall now consider a system of rectangular coordinates, such that the Y axis represents the plane of the plate and the X axis represents the neutral plane. The point p is produced by the tube at the upper level A; p' is produced by the tube at the lower level—minus A. The actual position of the indicator point is at the intersection of the line Ap and $-Ap'$. The equation of the line Ap is

$$\frac{y + p}{x} = \frac{A + p}{D},$$

D being the distance of the plate from the center of the tube. Similarly, the equation of the line—Ap' is

$$\frac{y + p'}{x} = \frac{-A + p'}{D}$$

Solving the foregoing equations as two simultaneous equations, one obtains

$$-y = \frac{A\,(p + p')}{2A + (p - p')}$$

and

$$x = \frac{D\,(p - p')}{2A + (p - p')}$$

x and y are the coordinates of the point of intersection of the lines Ap and $-Ap'$. This is the actual position of the indicator point.

Let x' and y' be the coordinates of the actual position of the foreign body, and b and b' the positions of the lower and upper shadows of the foreign body; then, by substituting b for p and b' for p' in the last two equations, one can solve for y' and x'.

Now, if the apex of the cornea is c millimeters behind the point of the indicator, then the foreign body is y—c—y′ behind the apex of the cornea, and x—x′ is the amount which the foreign body is temporal or nasal to the vertical meridian plane (temporal if x—x′ is positive; nasal, if it is negative).

The plane along which the tube has been shifted is represented, as already indicated, by the vertical lines on the roentgenograms. Let K be the distance of the shadow of the indicator point from this line, and L the distance of the shadow of the foreign body from this line. These distances can be measured from either of the two roentgenograms. Let z be the actual distance of the indicator point from this line, z' the actual distance of the foreign body from this line. Let T be the position of the tube center. Draw the lines TK and TL. Draw a line parallel to the Z axis at the distance x from it (which has already been determined), until it intersects the line TK. Draw a similar line at the distance x' from the Z axis (which has also been determined), until it intersects the line TL. From the two points of intersection draw perpendiculars to the Z axis. Then, from similar triangles,

$$z = K\left(1 - \frac{x}{D}\right)$$

and

$$z' = L\left(1 - \frac{x'}{D}\right)$$

$z—z'$ is the amount which the foreign body is above or below the horizontal meridian section of the eyeball. If z—z′ is positive, the foreign body is below this section; if it is negative, the foreign body is above this section.

LOCALIZATION IN THE HIGHLY AMETROPIC EYE

It is now time to drop my second assumption, that an emmetropic eye is being dealt with, and consider localization in the ametropic eye.

The rules and principles that have already been laid down in the consideration of borderline localizations will, in many cases, make further

investigation unnecessary. On the other hand, after applying the tests of increased precision, the need for considering the axial length of the eyeball may become more imperative.

Let *A* be the refractive error of an eye, expressed in diopters. The "convergence" (Gullstrand) of that eye is 58.64 + A; 58.64 being the convergence of an emmetropic eye, the focal length of that eye, in air, is

$$f' = \frac{1}{58.64 + A}$$

Its focal length in vitreous is *f*, which is $f' \times 1.336$. Its axial length is $f + 1.6$; 1.6 mm. being the distance of the posterior principal point of the eye from the apex of the cornea. This axial length is from the apex of the cornea to the posterior pole of the retina only. It is therefore

$$\frac{1.336}{58.64 + A} + 1.6 \text{ mm.}$$

Von Rohr has calculated, tabulated and expressed graphically the relation between the refractive error and the axial length of an eye in this manner. The results are given to the second decimal point and cover the range between + 10 and — 20 diopters. Since an accuracy of 0.5 mm. is sufficient for the purpose of localization of foreign bodies, I have derived from these tables the following simple rule for the rapid calculation of the axial length of an ametropic eye: The axial length of the axially emmetropic eye is 24.4 mm. If the eye is hyperopic, subtract 0.33 mm. for each diopter up to 10 diopters of refractive error. If the eye is myopic, add 0.5 mm. for each diopter of refractive error up to 10 diopters, 0.6 for each of the next 6 diopters and 0.75 for each of the last 4 diopters. Add 1.25 mm. for the thickness of the ocular wall at the pole.

Since the localization of ocular foreign bodies is made with reference to a system of rectangular coordinates which originate at the apex of the cornea, each coordinate being parallel to one of the principal planes of the eye, it becomes necessary to adjust the eye with reference to the x-ray plate accordingly. By adjusting the patient's head so that a line passing through the apexes of the two corneas is perpendicular to the plate, and fixing the vision on a point which is on the prolongation of the optic axis of the eye to be examined, the proper directions of the principal planes of the eye is insured.

This applies only in cases of orthophoria. In cases of heterophoria, the adjustment must be made accordingly. Since the affected eye is partly covered by the indicator, and since its sight is usually so impaired that it could not be used for fixation anyway, fixation is effected by the uninvolved eye. If the uninvolved eye is totally blind, exact localization is extremely difficult. If blepharospasm is present, it should be relieved before an accurate localization is attempted.

APPLICATION OF THE WESSELY KERATOMETER

Localization of the apex of the cornea is effected by centering the point of an indicator in front of it. For precise measurement of the distance between the cornea and the indicator point I use and recommend the Wessely keratometer. This instrument is ideally suited for the purpose. The ophthalmologist needs no description of it. The roentgenologist who does this kind of work is urged to acquire it and become proficient in its use.

SOME MODIFICATIONS OF THE VOGT (BONE-FREE) METHOD

A method which has recently come into use, which has a limited but indispensable application, is the bone-free roentgenography of the anterior segment of the eyeball, invented by Vogt. A small film of special shape is introduced into the anesthetized conjunctival sac nasal to the globe. The x-ray tube is centered perpendicularly to the film, over the apex of the cornea, from the temporal side. Five films are taken in this manner, the tube and film remaining in the same position, but the optic axis directed as follows: (1) straight forward, (2) nasally, (3) temporally, (4) upward, (5) downward. A similar set of five films is taken with the film below the eyeball and the tube above.

In the original method of taking these pictures a specially designed film holder is used, which is held by the assistant. I have found that the film can be inserted deeper and with less discomfort to the patient without a holder. I have also found it more convenient to let the patient hold the film in place after it has been properly adjusted. In the Vogt Clinic the films are identified by writing on the envelope and later transferring the legend to the film, before the latter is developed. I find it much easier and surer to attach a lead figure to each film packet in advance. The films are numbered from 1 to 10 and the order already given is rigidly adhered to, and is indicated on the report, or on the card on which the entire set is mounted. Sometimes it is necessary to make only a few of these roentgenograms. It is then more convenient to mount them between two glass lantern slide covers.

OPHTHALMOSCOPIC LOCALIZATION

So far I have discussed the method of localizing foreign bodies of the eye by the x-ray method. When the media are clear and the foreign body or its site can be seen with the ophthalmoscope, an accurate localization can be made by applying the principles of refraction. This method may be the only one possible to use in some cases, as when the foreign body is nonmetallic and is in the posterior segment of the eyeball. This method will also find application in the localization of tears of the retina, as becomes necessary before the Gonin operation for detached retina can

be performed. It may also be used for the localization of minute intraocular tumors, cysts, hemorrhages or other pathologic processes.

I have already suggested that a more satisfactory method of stating the location of a point in the eyeball is by giving the direction of the meridional section which passes through that point, and the location of the point in that section. Since the present problem involves consideration of optical principles, the meridional method is not one of choice, but of necessity.

I shall consider a meridional section of the Gullstrand schematic resting eye. A ray of light proceeding from a point on the retina toward the posterior nodal point will emerge in a parallel direction but passing through the anterior nodal point. As long as the emerging ray passes through the pupil, it will be visible to the observer.[2] I shall show what happens when the pupil is 5 mm. in diameter. By the graphic method it is seen at once that when the luminous point is 11 mm. from the pole of the retina the emergent ray just strikes the margin of the pupil. In order to see a point beyond that, one can no longer look through the anterior nodal point of the observed eye, but must look to one side of it. One shall naturally look along the line that gives the minimum deviation of the emergent ray and the maximum definition of the image of the point. The incident ray that fulfils these requirements is the one that strikes the posterior surface of the lens as near as possible to the posterior nodal point of the eye, and yet emerges through the pupil. Such a ray will emerge at the margin of the pupil. Generally speaking, the emergent ray will be so directed that a point observed will appear to be more anterior than it actually is, that is, the image will be displaced forward. The amount of displacement will be greater, the more anterior the location of the point observed. I have worked out graphically the position of the emergent ray which is produced by an incident ray proceeding from any point of the meridional section of the retina which is situated up to 5 mm. behind the limbus, measured along the retinal surface. The luminous points on the retina were assumed 1 mm. apart, measuring from the posterior pole, along the retinal surface. The results were tabulated and then plotted, the apparent position of a point as the ordinate and the actual position as abscissa.

The position of each point (real or apparent) has been given with reference to the posterior pole of the retina, the equator, the critical point (11 mm. from pole) and the limbus. The actual positions are also given in terms of the length of the cord from the limbus. It appeared from the graph that, within the practical limits, all the points were essentially on a straight line. From the equation of this line I derived a series of

2. The defects of the optical system, such as spherical aberration, chromatic aberration, deformation of oblique bundles, etc., have been neglected.

formulas which enables one to calculate rapidly the actual position of a point on the retina from its apparent position. The actual position is given in terms of millimeters from the limbus, measured along the periphery of the eyeball, and also along the cord. The apparent position is given in terms of millimeters, and also in disk diameters, from the pole, equator and orra serrata.

The method used to obtain these results follows: A meridional section of Gullstrand's schematic resting eye was drawn to a scale of 1 cm. to the millimeter from the data [3] in table 1. The anterior nodal point is 0.1 mm. in front of the posterior surface of the lens, or 7.1 mm. from the apex of the cornea; the posterior nodal point, being 0.25 mm. behind that, is 0.15 mm. behind the posterior surface of the lens, or 7.35 mm. from the apex of the cornea.

The anterior and posterior principal points of this optical system are situated 1.35 and 1.6 mm. respectively behind the apex of the cornea.

TABLE 1.—*Indexes of Refraction, Thickness and Curvatures of Refractive Media of Eye; Curvature of Retina*

Structure	Axial Thickness, Mm.	Radius of Anterior Surface, Mm.	Radius of Posterior Surface, Mm.	Index of Refraction
Cornea	0.5	7.7	6.8	1.376
Aqueous	3.1	6.8	10.0	1.336
Lens				
Anterior cortex	0.546	10.0	7.911	1.386
Equivalent nuclear lens	2.419	7.911	— 5.76	1.406
Posterior cortex	0.635	— 5.76	— 6.0	1.386
Vitreous	17.20	— 6.0	—11.4	1.336
Retina		—11.4		

The relative indexes of refraction applicable at the various refracting surfaces are, for a ray which proceeds from the retina forward: at posterior cortical surface of lens, 1.038; at posterior nuclear surface of lens, 1.015; at anterior nuclear surface of lens, 0.985; at anterior cortical surface of lens, 0.964; at posterior surface of cornea, 1.030; at anterior surface of cornea, 0.727.

In order to expedite the graphic work, a unit of 20 cm. was laid off on a straight line and the aforementioned quantities laid off on the same line to this scale (20 to 1). Thus 1.038 is represented on this line by 20.76 cm., 1.015 by 20.30 cm., etc.

For a ray of light entering the eye, the reciprocals of these values are used.

A point is now indicated on the anterior surface of the lens, 2.5 mm. below the optic axis. This represents the lower margin of the pupil at its intersection with the meridional plane. A line is drawn connecting

3. von Rohr: Eyes and Spectacles, Berlin, Julius Springer, 1912.

this point with any point of the retina above the optic axis. At the point where this line strikes the posterior surface of the lens the tangent is drawn to the surface. With the same point as a center and the unit as a radius an arc is described cutting this line behind the lens. Another arc is similarly drawn with 1.038 as a radius. The line and the two arcs are marked by the figure 1. From the point of intersection of the line with the first arc a perpendicular is drawn to the tangent just mentioned. This perpendicular is prolonged until it strikes the second arc. From this point a line is drawn to the first point of incidence and is continued to the posterior nuclear surface of the lens. This line is marked by the figure 2. At the second point of incidence the tangent is again drawn to the surface, and from this point two arcs are described with the appropriate radii (the unit, 20 cm. and the length representing the rela-

TABLE 2.—*Relation of Actual to Apparent Position of a Point on the Retina* *

Actual Position		Apparent Position		
Distance from Pole	Distance from Limbus	Distance from Pole	Distance from Limbus	Distance from Orra Serrata
12	18	12.4	17.6	13.1
13	17	14.0	16.0	9.5
14	16	15.15	14.85	8.35
15	15	16.5	13.5	7.0
16	14	18.0	12.0	5.5
17	13	19.2	10.8	4.3
18	12	20.6	9.4	2.9
19	11	22.25	7.75	1.25
20	10	23.5	6.5	0.0
21	9	25.1	4.9	—1.6
22	8	26.7	3.3	—3.2
23	7	28.25	1.75	—4.75
24	6	30.0	0.0	—6.5

* All distances are in millimeters.

tive index of refraction 1.015, hence 20.30 cm.), and the second refracted ray determined, which is marked with the figure 3. In this manner, the ray is traced until the emergent ray is obtained, which is indicated by the figure 7. This line is continued until it strikes the retina, which will take place in front of the incident ray. The amount of forward displacements is measured along the retina, and recorded (table 2; fig. 5).

The origin of coordinates of this graph, 0, corresponds to the critical point, which is 11 mm. from the pole, 19 mm. from the limbus and 12.5 mm. behind the orra serrata, all measured along the retinal surface.

The actual distance A_{1c} is measured along the cord from the limbus.

The equation of the broken line is

$$x = 0.6\,y + 1.6$$

If B_{os} is the estimated apparent distance from the orra serrata, then

$$y = 12.5 - B_{os}$$
$$x = 19.0 - A_{1c}$$

Substituting these values in the first equation and solving for A_{1c}, one obtains

$$A_{1c} = 9.9 + 6/10\ B_{os} \text{ or roughly}$$
$$A_{1c} = 0.6\ B_{os} + 10$$

If B_{os} is expressed in disk diameters, one has, since 1 disk diameter equals 1.5 mm:

$$A_{1c} = 0.9\ B_{os} + 10$$

From this the following rule is derived for the estimation of the actual position of a point on the retina when the apparent position is known:

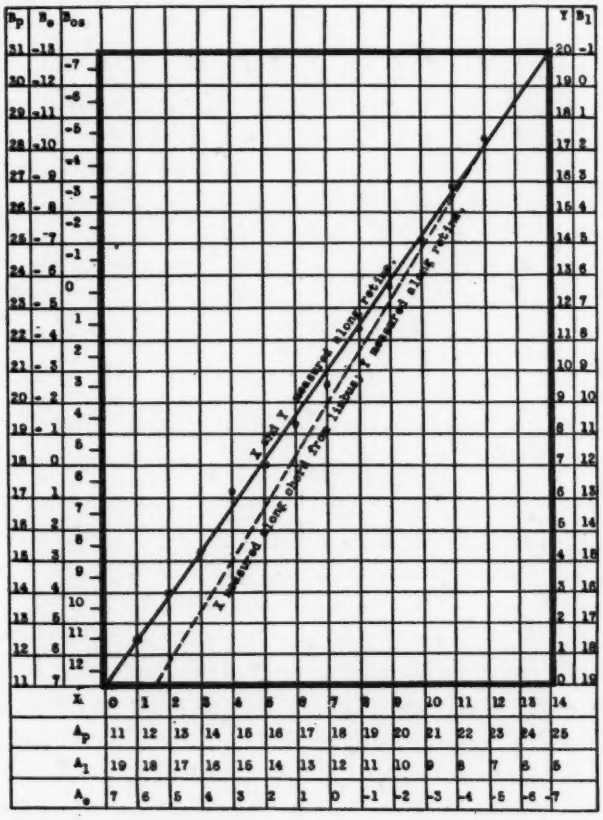

Fig. 5.—Graph showing relation of actual to apparent position of a point on the retina. All distances are in millimeters. The diameter of the pupil has been assumed as 5 mm. The relation differs with the size of the pupil. The actual positions have been plotted as abscissas and the apparent positions are ordinates. A_p and B_p, distances from pole; A_e and B_e, distances from equator; A_1 and B_1, distances from limbus; B_{os}, distance from orra serrata; X and Y, distances from critical point, which is 11 mm. from pole when the pupil is 5 mm. For larger pupil it is further from the pole, and for a smaller pupil it is nearer the pole.

Estimate the distance of a point in disk diameters from the periphery of the retina; multiply by 0.9 and add 10. The result is the actual distance in millimeters along the cord from the limbus.

This problem can be solved also mathematically, in the following manner: I shall consider a system of rectangular coordinates which originate at the apex of the cornea, as already described. A series of spherical surfaces are being dealt with. Their meridional sections are therefore arcs of circles. The general equation of a circle, if the coordinates of its center are b and c, and its radius is r, is

$$(y-b)^2+(z-c)^2=r^2$$

Since the centers of all the circles being dealing with are located on the Y (optic) axis, the equation reduces to the simpler form

$$(y-b)^2+z^2=r^2$$

By substituting the appropriate values of b and r, the equations of the various surfaces of the eyeball are derived. These are given in table 3.

TABLE 3.—*The Ocular Surfaces: Their Radii; Locations of Their Centers. Their Equations*

Surface	b (in Mm.)	r (in Mm.)	Equation	
Cornea, anterior	7.7	7.7	$(y-7.7)^2+z^2=7.7^2$	(A)
Cornea, posterior	7.3	6.8	$(y-7.3)^2+z^2=6.8^2$	(B)
Lens cortex, anterior	13.6	10.0	$(y-13.6)^2+z^2=10^2$	(C)
Lens nucleus, anterior	12.057	7.911	$(y-12.057)^2+z^2=7.911^2$	(D)
Lens nucleus, posterior	0.805	— 5.76	$(y-0.805)^2+z^2=5.76^2$	(E)
Lens cortex, posterior	1.20	— 6.0	$(y-1.20)^2+z^2=6.0^2$	(F)
Retina	13.0	—11.40	$(y-13.0)^2+z^2=11.4^2$	(G)
External surface of sclera	13.3	—12.35	$(y-13.3)^2+z^2=12.35^2$	(H)

If p is the radius of the pupil, then the location of its lower intersection with the meridional plane is

$$y=13.6-\sqrt{100-p^2}$$

z equals — p, which I shall represent by d. For a 5 mm. pupil, the location is (3.92), (— 2.5).

The general equation of a line passing through this point is

$$z+p=\tan\gamma\,(y-d),$$

where γ is the angle between the line and the optic axis. By solving this and (B) as two simultaneous equations, the coordinates of the point of intersection of this line are obtained with the posterior surface of the cornea. Dividing the z coordinate of this new point by 6.8 will give the sine of the angle γ' which the radius to this point makes with the optic axis. The angle of incidence at this point is the difference between

the two foregoing angles. If this is represented by $8''$, then one can solve for the direction of the ray in the substance of the cornea by means of the equation

$$1.030 \text{ sine } \beta'' = \text{sine } 8''$$

One can determine from this the angle β between the optic axis and this line. The equation of this line is solved with A as two simultaneous equations. Dividing the new z coordinate by 7.7 will give the sine of the angle β', which the radius to this point makes with the optic axis. The difference between the two latter angles is the angle of incidence of this ray. One can now solve for the direction of the emergent ray, remembering that at this surface the relative index of refraction is 0.727. One can represent the angle between the emergent ray and the optic axis by $\propto$. From the starting point (pupillary margin) one can now proceed in the opposite direction (toward the retina) and trace the ray through the three schematic layers of the lens and through the vitreous to the retina. The angle between the optic axis and the ray in the vitreous is then designated by Δ. It is obvious that for every value of $\propto$ there will be a corresponding value of Δ, provided the pupil remains constant, but that the relation between these angles depends on the size of the pupil.

A SIMPLIFIED APPARATUS

The process of localizing a foreign body consists of three steps: (1) obtaining the x-ray pictures; (2) determining from these the location of the foreign body by graphic triangulation and (3) indicating the position of the foreign body on a chart. I have already discussed all three of these, but I wish to add to the first two.

There are several special apparatus for taking roentgenograms of the orbits for localization of foreign bodies. They are termed "localizers," which they are not. A more descriptive name would be "picture takers" for that is all they are used for. The simplest of these, which has so far been described, is Dr. Dixon's modification of the Mackenzie Davidson apparatus. Having obtained from Dr. Dixon permission to build one of these, in September, 1924, I simplified it still further by elimination of the rack-and-pinion method of shifting the tube. I mounted the tube holder on four telescoping posts. A wooden block placed under the tube holder fixed the level of the tube. The dimensions of this block were such that three fixed positions could be obtained: one neutral (center of tube at level of horizontal wire) and one each 3 cm. above and below this level. A spirit level was placed on the platform bearing the tube, so that the tube would be shifted in a truly vertical plane. In 1927, I built another apparatus. This, which is still in use, consists of a separate tube-holder and plate-holder, both of which can be hung on the wall when not in use, thus saving space and reduc-

ing the chances of breaking the tube. The base of the tube holder is provided with four posts which support the tube platform at a level which brings the tube center 3.0 cm. below the neutral plane. The platform is provided with two legs which telescope between the four posts of the base. Each of these legs is provided with two horizontal spring-actuated plungers. These are fixed at such a height that when

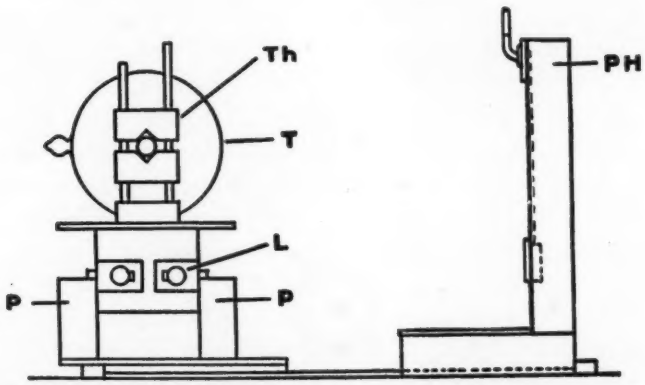

Fig. 6.—Side elevation of simplified apparatus; *P*, guide posts; *L*, automatic height locks; *Th*, tube holding clamps; *T*, x-ray tube; *PH*, plate holder.

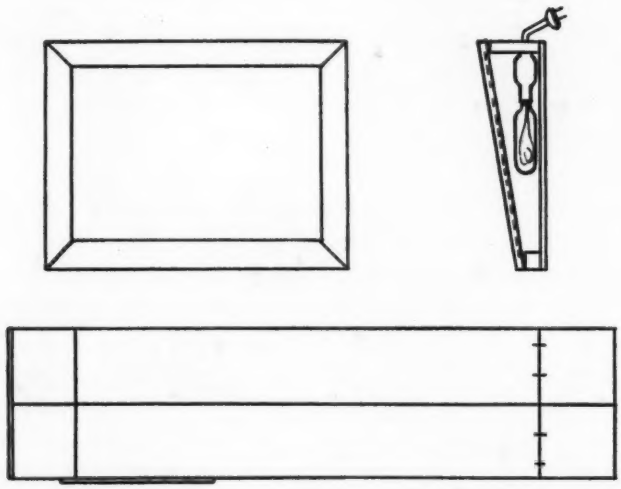

Fig. 7.—The upper figure shows the top and side views of a special viewing box for localization of a foreign body; the lower figure is the top view of the special drawing board.

the tube platform is raised to 6.0 cm. above the lowest position, the plungers are automatically released, thus fixing the center of the tube at a level 3 cm. above the neutral plane. Another set of plungers will automatically stop the tube at the neutral level.

The plate holder is an L-shaped contrivance consisting of a horizontal platform for the patient's head and a vertical frame for holding the plate. The letters *R* and *L* are attached to this frame in such a manner that they indicate automatically which side of the head was near the plate. A lead marker bearing the owner's name is also permanently fixed in the frame. The apparatus can be set up in about thirty seconds.

A CONVENIENT DRAWING BOARD

I have devised a convenient small drawing board for localizing the foreign body from the roentgenograms. The board is 6 inches (15.24 cm.) wide and 24 inches (60.96 cm.) long. The working surface is painted white. A horizontal line (parallel to the long edge) indicates the neutral plane. A vertical line at the left indicates the plane of the film. At right, 47.5 cm. from this line, is another vertical line representing the plane in which the tube center is shifted. This line is crossed 3 and 6 cm. above and below the neutral level, these points indicating the tube positions to single and double scale. A narrow strip of wood is nailed to the left vertical edge, rising slightly above the working surface. A similar strip is nailed to the lower edge. These provide guides for the triangles, so that horizontal and vertical lines can be drawn without the use of a T square. Transparent or translucent paper is used for drawing, the necessity of drawing all the basic lines and points each time is avoided. In addition to these lines there is a list of magnetic and diamagnetic substances printed on the board, so that one can tell whether the foreign body will react to the giant magnet if one knows its substance.

A CONVENIENT VIEWING BOX

The use of an ordinary viewing box while measuring the distances of the pointer and foreign body from the principal lines is very inconvenient. The ideal place for the film or plate is on the table, next to the drafting board. I have devised for this purpose a small horizontal viewing box, with a flashed opal glass 7 by 10 inches, so that it just holds two 5 by 7 inch films. A single 15 watt tubular bulb of the candelabra type illuminates the two films. As the blackest parts of the films cover the center of the glass, there is no glare when the box is in use. The top is hinged, so that both sides of the glass can be cleaned. A switch is provided at the left end. The inside surfaces are not painted, but a sheet of white paper is inserted for reflecting the light; it is easier to insert a fresh piece of paper than to keep a painted or enameled surface clean. Sometimes the use of green, yellow or blue paper brings out more details when the foreign body is very small, or its shadow is faint.

The board and viewing box can be built as one unit. An eyelet is provided for hanging these on the wall.

A very fine pair of bow dividers are used for transferring the distances from the roentgenograms to the paper. For measuring, I use a small translucent combination scale-and-slide-rule, so that the calculations can be made rapidly after the measurements have been determined. In borderline cases the localization is checked up on a 10:1 meridional section of the eyeball.

SUMMARY

The true location of a foreign body cannot be indicated on the present charts (which are orthographic projections), except when the foreign body happens to be in the principal horizontal or vertical section. This applies also to its relation to the structures of the eye or orbit. By indicating the foreign body on the meridional section which passes through that foreign body, one can get not only the exact location of the foreign body, but its relation to the structures of the eye or orbit.

I have designed an improved chart, called the meridional localization chart, which has been combined with the present orthographic chart, so that both forms of localization can be made on the same drawing.

If a point of a foreign body is *a*, *b*, and *c'* from the principal vertical, horizontal and equatorial sections, respectively, then its distance from the center of the eyeball is

$$\sqrt{a^2 + b^2 + c'^2}$$

If the result is less than 12.4 mm., the foreign body is intra-ocular. If it is more than 12.4 mm., the foreign body is extra-ocular. If it is between 11.4 and 12.4 mm., the foreign body is in the ocular wall. When the foreign body is less than 3.6 mm. behind the apex of the cornea, special application of this formula will indicate whether it is intra-ocular; and, if intra-ocular, whether it is in the lens, posterior chamber, etc.

If the two ends of a foreign body have been localized, then its length can be calculated in the following manner: Subtract the three coordinates of one end from the corresponding three of the other end. Add the squares of the differences and extract the square root of the sum.

When the localization shows the foreign body to be on the borderline between intra-ocular and extra-ocular then (provided it has not been disturbed by the magnet), as a general rule, its direction will indicate its location; if it is parallel to the ocular wall, it is on the retina; if it is perpendicular to the ocular wall, it is in the wall. If it is neither parallel nor perpendicular, it is probably extra-ocular, except when it

is in the anterior segment. In the latter condition similar reasoning will indicate whether the foreign body is in the ciliary body, lens, etc.

When localizing by the method of graphic triangulation, the precision can be increased by doubling or tripling the vertical distances but retaining the 1 : 1 scale for the horizontal distances.

Algebraic calculation can be used to supplant or supplement the graphic method. The formulas are given in the text.

When localizing in the highly ametropic eye due allowance must be made for the length of the eyeball. From the calculations of von Rohr I have derived a simple rule for rapidly estimating the axial length of an eye of known refractive power: The emmetropic eye is 24.4 mm. long; subtract 0.33 mm. for each diopter of hyperopia (up to 10 diopters); add 0.5 mm. for each of the first 10 diopters of myopia, 0.6 for each of the next 6, and 0.75 for each of the next 4. Add 1.25 mm. for the thickness of the ocular wall at the pole.

The distance between the lead indicator and the apex of the corneas can be measured by means of the Wessely keratometer.

By means of the Vogt bone-free method of ocular roentgenography extremely small or nonmetallic foreign bodies can be demonstrated. Some modifications have been described in the text. I have used it also to demonstrate foreign bodies in the lids.

The following simple apparatus for localizing ocular foreign bodies has been described: a simplified "localizer," a convenient drawing board and a convenient viewing box.

OCULAR SYMPTOMS IN THE DIAGNOSIS OF TUMOR OF THE BRAIN*

WALTER D. ABBOTT, M.D.
DES MOINES, IOWA

The progress made in surgical procedures on the brain during the past two decades has not been due to any one factor, but to refinements in diagnosis and technic and, most of all, to the cooperation of neurologists, neurosurgeons, internists and ophthalmologists.

If further strides are to be made, one must have closer cooperation and be ever on the alert to attach significance to early symptoms. Tumors of the brain are malignant in view of their appalling mortality, and experience has taught that the only successful means of dealing with malignant tumors is through early diagnosis and treatment.

The cardinal symptoms leading to a diagnosis of tumor of the brain are (1) headache, (2) choked disks and (3) vomiting; however, one or more of these indications may be conspicuously absent. The importance of a thorough ophthalmologic examination in a case of suspected tumor of the brain cannot be estimated because of the frequency with which the ocular symptoms are of definite localizing value. The manifestation of involvement of the optic tract may appear at any place along the course of the visual pathway, and an attempt will be made to present the various types of lesions and their clinical significance.

ANATOMY

A knowledge of the anatomic conditions governing the course of the optic pathway enables one to determine precisely the importance of a focal lesion. From the retina the optic fibers pass, in the optic nerve trunk, to the optic chiasm. Here they undergo a partial decussation; that is, the fibers from the right halves of the two retinas continue in a single trunk on that side, and fibers from the two left halves of the retinas do likewise on the left side. Each optic tract continues in a dorsal direction, winding around the cerebral peduncle, and ends in the primary visual centers on the same side. These centers are the posterior portion of the optic thalamus (pulvinar), the lateral geniculate body and the superior quadrigeminal body. From these centers the optic stimuli are conveyed in two directions: 1. From the central visual tract (Gratiolet's bundle) they pass to the cortical visual area of the same

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* Read before the Des Moines Academy of Ophthalmology and Oto-Laryngology, Jan. 19, 1931.

side on the mesial surface of the occipital lobe. This area is known as the cuneus, and from here association fibers pass to the lateral surfaces of the occipital and parietal lobes (centers of visual memory). 2. From the superior quadrigeminal body, nerve fibers course to the small cell lateral nucleus (Edinger-Westphal) of the oculomotor nerve. This tract carries the impulses for the reflex pupillary arc (chart 1). This connection is both crossed and homolateral.

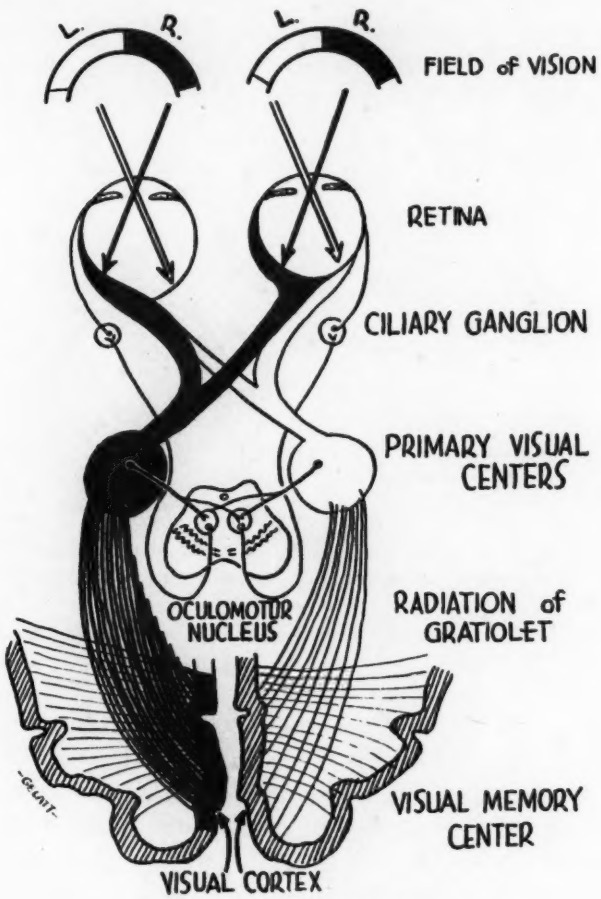

Chart 1.—The optic pathway.

LOCALIZATION OF LESIONS OF THE OPTIC TRACT

The appearance of choked disk or atrophy of the nerve heads is not indicative of focal lesions, but may be caused by pressure exerted at any place within the cranial cavity. In describing the various lesions, reference will be made to the conditions of the nerve head and the presence or absence of the pupillary reflex as may be indicated by any deviation from the normal.

1. *Lesions of the Optic Nerve.*—Total destruction of the optic nerve at a point peripheral to the chiasm will result in complete blindness of one eye and absence of the pupillary reflex. If only a portion of the optic fibers is destroyed, scotomas or narrowing of the field of vision will be encountered instead of amaurosis. A lesion in this region produces the classic and diagnostic syndrome described by Foster Kennedy,[1] known as the basal frontal syndrome. This characteristic clinical picture is seen in cases of tumor of the olfactory groove and shows loss of central acuity, a central scotoma and a normal, pale or choked disk on the homolateral side. The contralateral eye has normal visual acuity, choked disk and a normal field of concentric contraction. The accompanying symptoms are headache, vomiting, stupor and loss or diminution of the sense of smell on the affected side (chart 2).

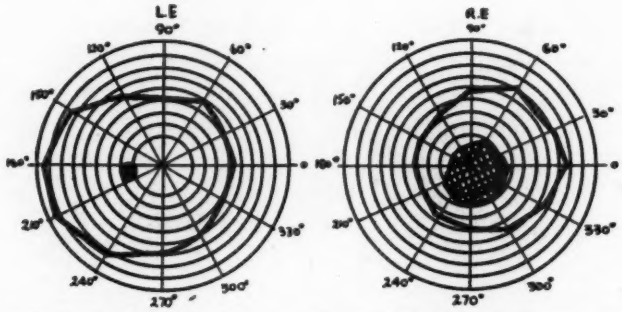

Chart 2.—Perimetric fields in a case of right basal frontal tumor. This patient had a central scotoma, diminished visual acuity, concentric contraction of the visual field and pallor of the disk of the homolateral eye. The contralateral eye had choked disk, concentric contraction of the visual field and normal visual acuity.

In cases of bilateral basal frontal tumor, what has been said for the homolateral eye now holds true for both eyes. Tumors in the frontal lobe, other than basal, do not cause such a characteristic ocular picture, and only about 40 per cent show choked disks early; the fields are normal or concentrically contracted.

A typical change in personality is observed in a large percentage of patients with tumors of the frontal lobe, whether the lesion is cortical, subcortical or basal.

Not infrequently a slow, irregular nystagmus is present in lesions of the frontal lobe, and this may be confusing when an attempt is made to distinguish between a frontal or cerebellar type of nystagmus. It is believed that there is a cortical center for the control and coordina-

1. Kennedy, Foster: Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes, Am. J. M. Sc. **142**:358, 1911.

tion of ocular movements, and when this inhibition is removed, the nystagmus appears in an irregular manner. The true cerebellar nystagmus is more rapid and regular. Thus careful study will usually enable the observer to distinguish between these two types.

2. *Lesions of the Chiasm.*—If a lesion involves only the mesial portion of the chiasm, as in tumor of the hypophysis, hydrocephalus or

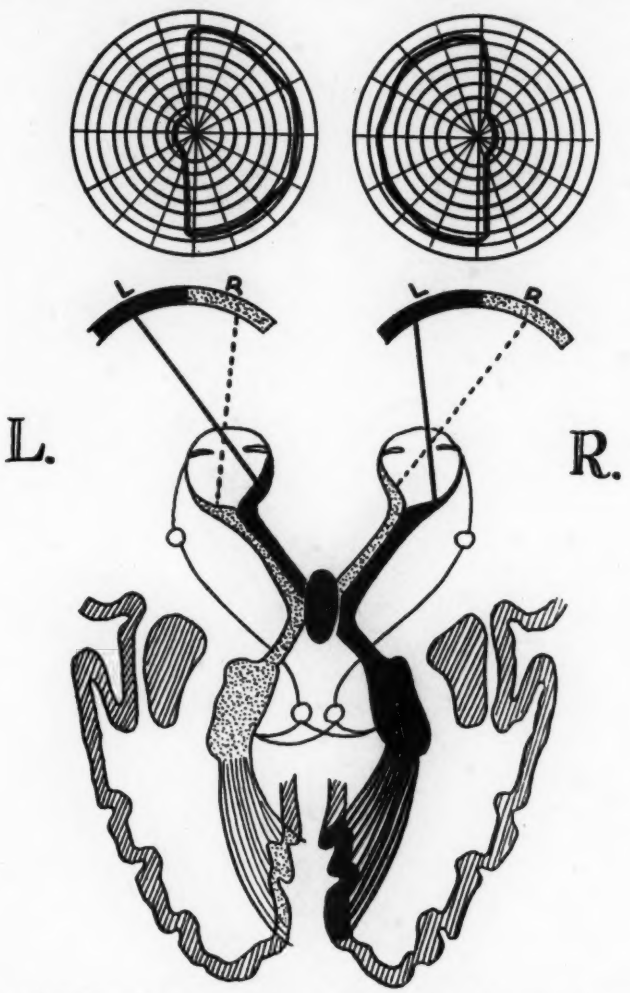

Chart 3.—A lesion of the mesial portion of the chiasm with a bitemporal hemianopia.

tumor of the third ventricle or tumor of the infundibulum, it destroys the decussating fibers that come from the nasal portions of the retinas, and the result is a bitemporal hemianopia (chart 3).

If, however, the external portions of the optic decussation are destroyed, an unusual condition is the result: a binasal hemianopia.

This rare phenomenon can be produced by a tumor of the third ventricle, aneurysm of both carotid arteries, symmetrical gummas, or possibly a cyst of the hypophysis bulging laterally (chart 4).

A lesion anterior to or on the lateral side of the decussation can produce blindness in one eye and temporal hemianopia (chart 5).

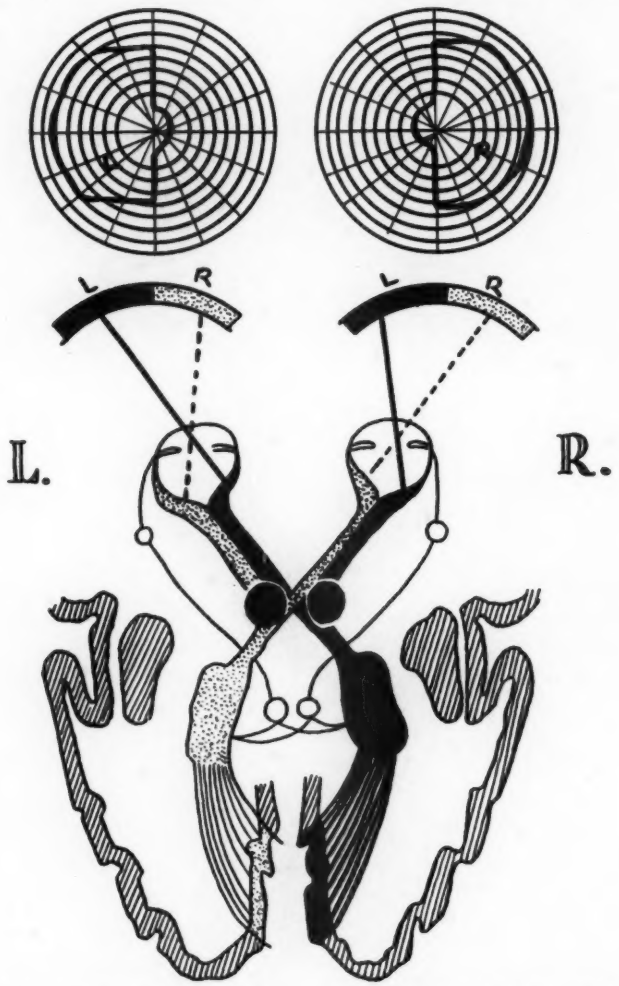

Chart 4.—Lesions of external portions of the chiasm with binasal hemianopia.

Homonymous defects (partial or complete) are the result of a lesion situated at the posterolateral side of the chiasm (chart 6).

The ocular symptoms accompanying lesions of the chiasm are: lowered visual acuity; normal or full pale disk; if of long standing, simple optic atrophy, and possibly a change in the pupillary reflex. The attendant neurologic symptoms are: headache (bitemporal or bifrontal); an increase in weight; a lowered basal metabolic rate;

polydipsia and polyuria; increased tolerance for sugar; cessation of the menses, and, in 32 per cent of cases of acromegalia, enlargement affecting the chiasm.

3. *Lesions of the Optic Tract, Primary Visual Centers and Radiation of Gratiolet.*—All of these lesions cause homonymous lateral hemianopia,

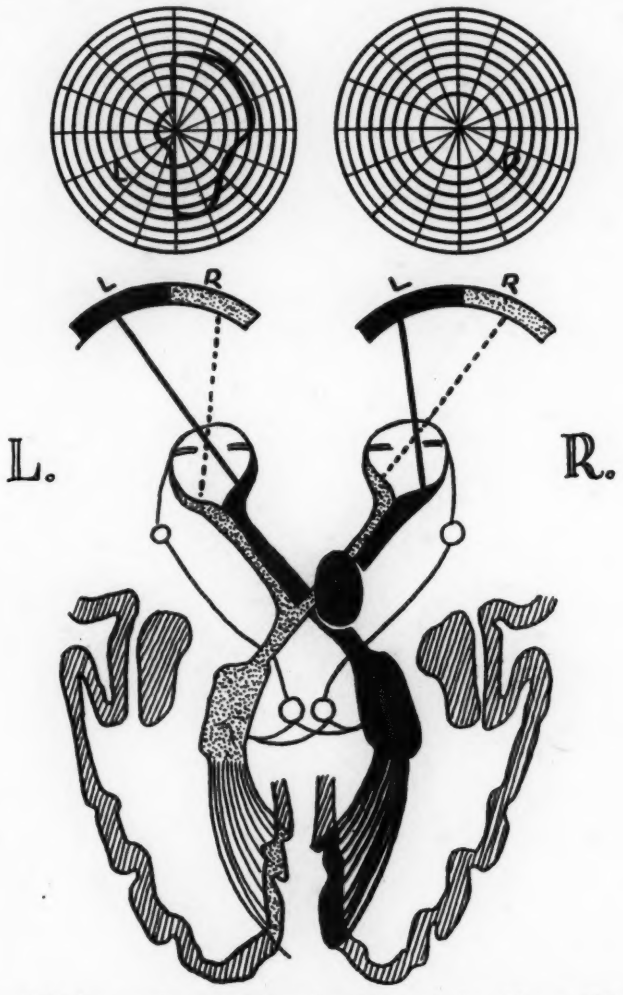

Chart 5.—A lesion on the anterolateral surface of the chiasm with blindness in the homolateral eye and temporal hemianopia in the contralateral eye.

because either both right or both left halves of the visual fields are involved (chart 7). If the lesion is peripheral to the point at which the reflex fibers to the Edinger-Westphal nucleus are given off, a ray of light thrown on the blinded halves of the retinas does not cause pupillary contraction, because there is no access to the oculomotor nucleus. If, however, the lesion is central to the point at which the reflex fibers

are given off, illumination of the blind halves of the retinas will cause contraction of the pupil because the stimulus, although it does not reach the cortical visual centers, reaches the centers for the pupillary sphincters. This hemianopic pupillary reaction is known as Wernicke's phenomenon.

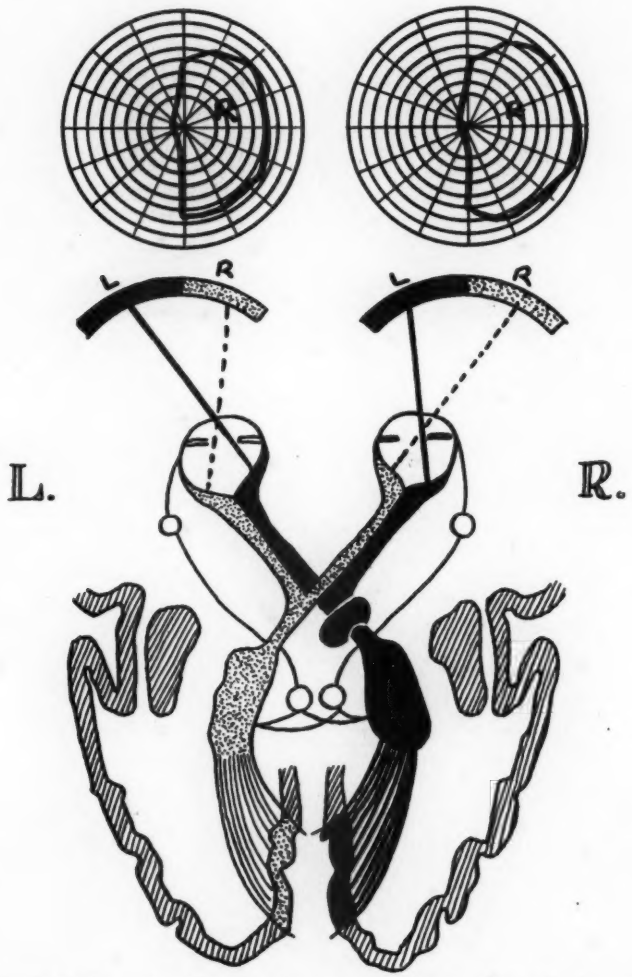

Chart 6.—A lesion situated at the posterolateral side of the chiasm with homonymous hemianopia.

Bilateral lesions that involve the visual memory centers in the cortex or the association paths from the striated area cause conceptual blindness. The patient is able to see an object as a flat surface, but cannot state its type or use.

If the cuneus is destroyed in one hemisphere and the occipital convexity in the other is involved, conceptual blindness is superimposed on a homonymous hemianopia.

As all lesions along the course of the optic tract dorsal to the primary visual centers manifest a quadrant or complete homonymous hemianopia, and as the other ocular symptoms vary with the location of the lesion, it is felt that the tables will concisely illustrate the salient diagnostic features.

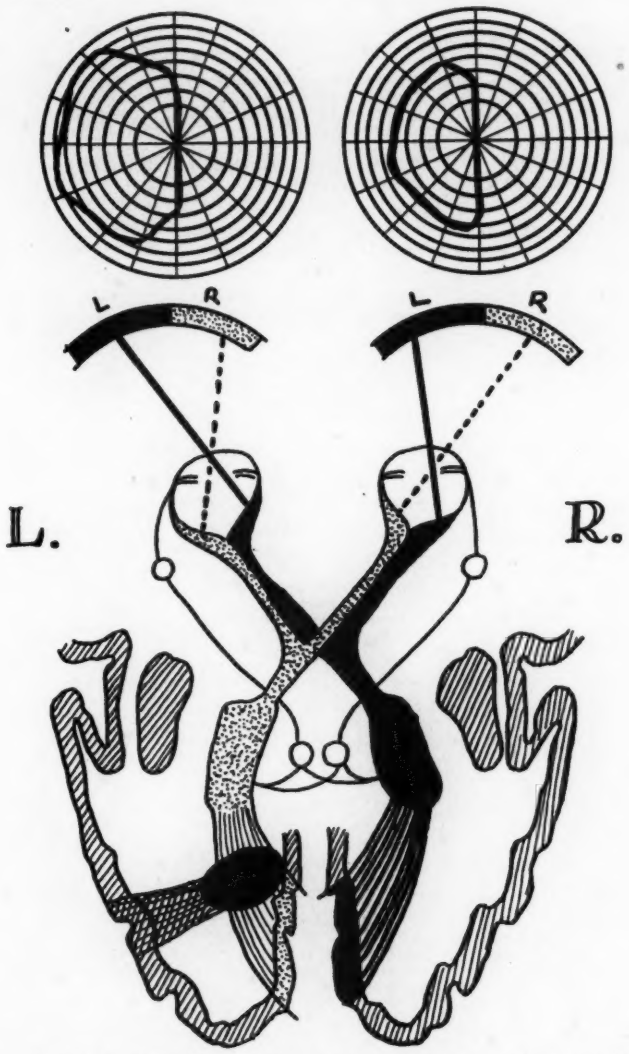

Chart 7.—A lesion posterior to the primary visual center with homonymous hemianopia.

From the accompanying charts and tables it would seem that the diagnosis of tumor of the brain is clearcut and obvious. However, it must be remembered that there are frequent deviations from the hard and fast rule, particularly with reference to lesions of the nervous system. The appearance of the optic disk may vary from slight hazi-

TABLE 1.—*Lesions of the Pons, Midbrain, Cerebellum and Medulla*

Location	Type	Neurologic	Ophthalmologic
Pineal gland	Frankl Hochwart	General signs of pressure; bilateral deafness; ataxia; precocity; indirect hypopituitary signs due to pressure	Bilateral choked disk; limitation of upward gaze normal or concentrically contracted fields
Third nerve nucleus	Nothnagel's syndrome	Ataxia; general signs of pressure	Bilateral choked disk; bilateral partial paralysis of third nerve; normal fields
Cerebellar tumor	Brun's syndrome	Ataxia; incoordination; general signs of pressure; ataxia before involvement of third nerve	Bilateral choked disk; bilateral partial paralysis of third nerve; normal fields; nsytagmus
Red nucleus	Benedikt's syndrome	Contralateral ataxia and incoordination	Bilateral choked disk; homolateral complete paralysis of third nerve
Cerebral peduncle	Weber's syndrome	Contralateral hemiplegia; general signs of pressure	Bilateral choked disk; homolateral complete paralysis of third nerve
Pons at level of sixth nerve	deRaymond's syndrome	Contralateral hemiplegia; general signs of pressure	Bilateral choked disk; homolateral paralysis of sixth nerve
Pons at level of sixth and seventh nerves	Millard-Gubler	Contralateral hemiplegia; general signs of pressure; homolateral complete paralysis of seventh nerve	Bilateral choked disk; homolateral paralysis of sixth nerve
Pons at level of seventh nerve	Foville's syndrome	Contralateral hemiplegia; homolateral complete paralysis of seventh nerve; general signs of pressure	Bilateral choked disk
Cerebellopontile angle	Angle syndrome	Homolateral deafness and tinnitus; paresthesia of fifth nerve (homolateral); later motor paralysis; complete paralysis of seventh nerve (homolateral); ataxia; general signs of pressure	Bilateral choked disk (50 per cent); anesthetic cornea (homolateral); paralysis of sixth nerve (homolateral); nystagmus

TABLE 2.—*Lesions of the Temporal, Parietal and Occipital Lobes and the Basal Ganglion*

Location	Type	Neurologic	Ophthalmologic
Temporal lobe	Tumor	Uncinate attacks and generalized convulsions; hemiparesis (contralateral); general signs of pressure	Bilateral choked disk; quadrant homonymous hemianopia; visual hallucinations (occasional)
Parietal lobe	Tumor	Incomplete sensory loss (contralateral); general signs of pressure	Bilateral choked disk; homonymous hemianopia (rare)
Occipital lobe	Tumor	Localized occipital headache; negative neurologic signs	Bilateral choked disk; homonymous hemianopia
Basal ganglion	Tumor of striate body	Parkinsonian syndrome	Bilateral choked disk; homonymous hemianopia
	Tumor of third ventricle extending laterally into thalamus and internal capsule	Fluctuating symptoms; general signs of pressure; contralateral hemiplegia and hemianesthesia; stupor; occasional convulsions	Normal, pale or choked disk; homonymous hemianopia; may have limitation of upward gaze

ness of the margin to a frank edema, and careful study is necessary to interpret correctly the importance of a departure from normal. Pressure is often capricious in causing a galaxy of symptoms, and is responsible for much confusion. I have operated in two cases of frontal tumor with partial paralysis of the third nerve which disappeared after decompression.

Thus it is necessary to correlate ophthalmologic, neurosurgical and general symptoms in a deliberate manner before a definite localization of cerebral neoplasm can be made.

820 Equitable Building.

UNILATERAL PIGMENTARY RETINITIS *

M. N. BEIGELMAN, M.D.
LOS ANGELES

Among various diseases of the retina, pigmentary degeneration was always considered one of the best outlined clinical entities. The ophthalmoscopic appearance of this disease, the changes in the visual fields, the state of adaptation and even the histopathologic observations, whenever they were available, presented little difficulty in diagnostic interpretation. However, with the introduction of finer methods of clinical investigation and with the accumulation of larger clinical experience, some atypical and rudimentary forms of pigmentary retinitis became known. Then new problems requiring further investigation came into existence.

An unusual variation of pigmentary retinitis, its unilateral form, was first reported as early as 1865 (Pedraglia [1]). So rare, however, was the unilateral occurrence of this disease that fifty years after the first report Leber [2] could find only fourteen similar cases in the literature. Of these, several were excluded because they seemed unreliable.

It is not surprising under these circumstances that doubts have repeatedly been voiced as to the very existence of unilateral pigmentary retinitis. Comments to this effect can be found in the writings of Schmidt-Rimpler,[3] Hutchinson,[4] Swanzy,[5] Guaita [6] and others. Shoemaker,[7] in his monograph on pigmentary retinitis, also took a negative stand in this problem, as expressed in the closing statement of his book: "unilateral retinitis pigmentosa is inconsistent with the author's ideas of the nature of the process."

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* Submitted for publication, March 4, 1931.

1. Pedraglia: Klin. Monatsbl. f. Augenh. **3**:114, 1865.

2. Leber, T.: Die Krankheiten der Netzhaut, Leipzig, Wilhelm Engelmann, 1916.

3. Schmidt-Rimpler: Der Augenheilkunde und Ophthalmoskopie, Braunschweig, F. Wreden, 1885, p. 277; cited by Günsberg: Ueber einen Fall von typischer Retinitis pigmentosa unilateralis, Arch. f. Augenh. **21**:184, 1890.

4. Hutchinson, J.: Suggestions for Clinical Work in Ophthalmology, Ophth. Hosp. Rep. **7**:431, 1873.

5. Swanzy: Dublin Quart. J. M. Sc. **51**:290, 1871; cited by Günsburg: Arch. f. Augenh. **21**:184, 1890.

6. Guaita: Recueil ophthalmologique, 1884, p. 394; cited by Gonin: Nouvelles observations de scotoma annulaire dans la dégénérescence pigmentaire de la rétine, Ann. d'ocul. **128**:90, 1902.

7. Shoemaker, W. J.: Retinitis Pigmentosa, Philadelphia, J. B. Lippincott Company, 1909.

Two objections are raised against cases reported as unilateral pigmentary retinitis: (1) the ophthalmoscopically and functionally normal eye might be affected in the future, particularly when the diagnosis is made on the basis of a single examination, or (2) the condition is not a true pigmentary retinitis but an acquired chorioretinal atrophy with pigmentation. Syphilis is often blamed. As Shoemaker put it: "The presumption of a syphilitic cause in unilateral cases is more justifiable than the diagnosis of retinitis pigmentosa."

When first confronted with a case of unilateral pigmentary retinitis, in 1925, I made an effort to clarify the nature of this exceptional occurrence by observation of the patient's condition for a period of several years.

REPORT OF A CASE

Mrs. R. S., aged 36, consulted me first in January, 1925. Her right eye was normal in all respects. The left one had a visual acuity of 20/25 with the correction of a moderate myopic astigmatism. The media were clear. Ophthalmoscopic examination of this eye revealed the following changes: a small number of pigment spots in the form of bone corpuscles, distributed at the periphery of the fundus, in some places along the retinal blood vessels; pallor and a waxy appearance of the optic disk; narrow retinal arteries; veins of normal caliber, and a concentrically limited visual field (fig. 1). The adaptation of this eye was seemingly lowered, but no quantitative test was made on this examination. The family history as to ocular lesions was negative. On general examination, no evidence of a systemic disorder was found.

The patient consented to remain under observation and was subsequently examined about once in six months. The right eye remained without change. The fundus and the visual functions, including that of adaptation to light, were normal. On the contrary, the pigmentation of the left retina and, to a lesser degree, the atrophy of the optic nerve were definitely progressing. In December, 1930, on examination at the Good Hope Clinic, the visual acuity of the left eye was 20/40 with correction. The visual field was contracted concentrically to about 10 degrees, with a relative scotoma in its temporal half (fig. 2). The adaptation to light, tested with the Birch-Hirschfeld apparatus, was one eighth of the normal limit. The pigmentation was considerably heavier than on first examination, and was still in the form of bone corpuscles. It appeared to approach closely both the optic disk and the macula (fig. 3). A posterior cortical cataract had been developing slowly in this eye during the last two years. The Wassermann test of the blood and urinalysis gave negative results.

In summarizing the case, one can say that a patient, aged 36, presented the following changes in the fundus of the left eye: deposits of pigment, mostly in the form of bone corpuscles, situated at the periphery of the retina; a waxy-yellow optic disk, and narrow arteries. The visual field of this eye was greatly contracted and the adaptation to light was lowered. This condition progressed markedly in the next six years, while the unaffected eye remained entirely normal.

Should this case be classified as a true pigmentary degeneration of the retina?

Both syphilis and trauma are known to be responsible for retinal atrophy with pigmentation, particularly when the condition is unilateral. Although no evidence of either syphilis or trauma was present in my case, an attempt to clarify this matter in general is justified because of the confusion existing in the literature with regard to the differential diagnosis.

In syphilis one usually sees chorioretinitis pigmentosa. As the name of the disease suggests, some signs of uveal involvement (vitreous

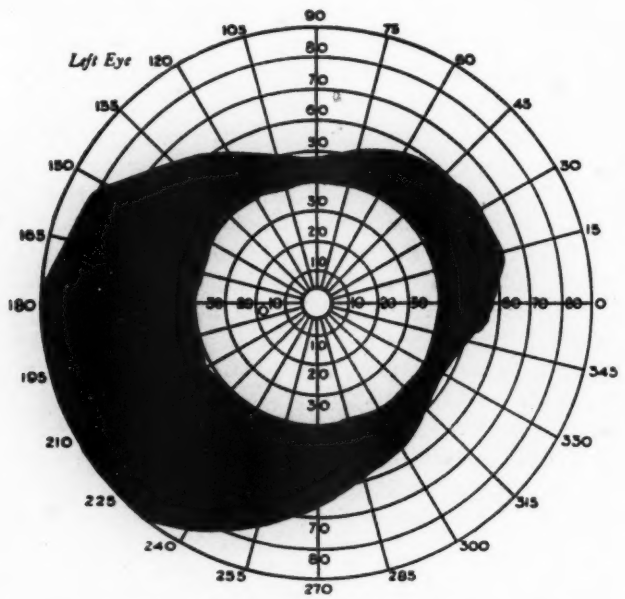

Fig. 1.—Visual field of left eye in January, 1925.

opacities, recent and old choroidal patches and even iridal adhesions) are not infrequently seen in these cases. In the retina, as Igersheimer [8] pointed out, vascular changes in the form of periarteritis and endarteritis contribute specific evidences of syphilis. The pigment is of a different type and localization from that in genuine pigmentary retinitis: it is distributed irregularly over the fundus, in clumsy black spots. The course of a syphilitic chorioretinitis also presents some characteristic peculiarities. The impairment of central visual acuity often progresses more rapidly than the peripheral contraction of the visual fields. Complications such as ocular palsies and acute iritis are likely to occur. Antisphilitic treatment will meet at times with favorable response.

8. Igersheimer, J.: Syphilis und Auge, Berlin, Julius Springer, 1928.

If some of these features are encountered in a case of retinal atrophy with pigmentation, the diagnosis of genuine pigmentary retinitis becomes most doubtful. On the contrary, if the changes in the fundus, the perimetric observations, the state of adaptation and the clinical course are characteristic of pigmentary retinitis, the latter diagnosis is not affected by a positive Wassermann reaction of the blood or by any other evidence of systemic syphilis.

Without taking a definite attitude as to the etiologic significance of syphilis in pigmentary retinitis, it must be stated that the simultaneous occurrence of both diseases is established not only by unquestionable

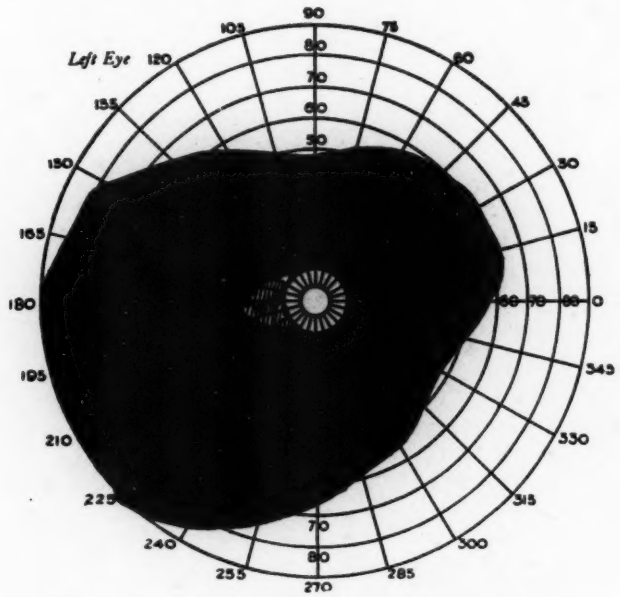

Fig. 2.—Visual field of left eye in December, 1930.

clinical observations, but by at least one pathologic examination. Friedenwald,[9] in the discussion of Verhoeff's paper at the sixty-sixth meeting of the American Ophthalmological Society, reported a case of typical pigmentary retinitis associated with a positive Wassermann reaction. A pathologic examination of both eyes revealed changes characteristic of pigmentary retinitis, without any evidence of syphilis either in the choroid or in the retina.

For these reasons I did not exclude from the group of unilateral pigmentary retinitis (table 1) the few cases with any evidence or sug-

9. Friedenwald, J., in discussion of Verhoeff: Microscopic Observations in a Case of Retinitis Pigmentosa, Arch. Ophth. **4**:767 (Nov.) 1930.

gestion of syphilis, so long as the clinical picture and course of the ocular disease were characteristic of pigmentary degeneration.

From the time of Wagenmann's [10] experiments (1865) in which severing of the posterior ciliary arteries resulted in retinal atrophy with pigmentation, the possibility of a traumatic origin of pigmentary retinitis, especially in its unilateral form, has been considered on several occasions. In the presentation of a case of monocular retinitis pigmentosa by Hine,[11] the discussion centered around the traumatic origin of pigmentary retinitis because of the history of failing vision since

TABLE 1.—*Summary of Observations in Cases of Unilateral Pigmentary Retinitis (Continued in Table 2)*

Case	Author	Year	Sex	Age	Eye Affected	Visual Acuity	Visual Field
1	Pedraglia (Klin. Monatsbl. f. Augenh. **3**: 114, 1865)	1865	M	36	Right	Snellen 111	Considerable concentric limitation
2	de Wecker (Traité de maladies d'oeil, 1868, vol. 2, p. 337)	1870	F	15	Left	1/00	.....
3	Baumeister (Arch. f. Ophth. **19**: 261, 1873	1873	M	44	Left	0	.....
4	Dehrig (Ueber Retinitis Pigmentosa, Bonn, 1882)	1882	F	46	Right	0.1	Markedly constricted
5	Günsburg (Arch. f. Augenh. **21**: 184, 1890)	1890	M	42	Left	.....	Constricted concentrically to about 15 degrees around fixation point
6	Gonin (Ann. d'ocul. **128**: 90, 1902)	1902	M	25	Left	.....	Nasal scotoma increasing with decrease of illumination; visual fields for red and blue coincide
7	Nettleship (Roy. London Ophth. Hosp. Rep. **17**: 1, 1907-1908)	1907	F	30	Right	6/18	.....
8	Hine (Proc. Roy. Soc. Med. [Sect. Ophth.] **17**: 14, 1924)	1924	M	34	Right	6/9	Field contracted to 5 degree circle
9	Rossi (Boll. d'ocul. **5**: 363, 1926)	1926	F	..	Right	5/5	Concentric limitation
10	Shoji (Arch. d'opht. **43**: 402, 1926)	1926	F	10	Left	0.5	Concentric limitation
11	Beigelman	1931	F	36	Left	20/25	Concentric limitation

injury to the affected eye. It was then brought out by A. L. Whitehead and by M. H. Whiting that neither of them had observed a bone corpuscle arrangement of pigment in the vast material of direct and indirect ocular injuries during the World War. It can be added that in the general ophthalmic literature there is not a single convincing instance of pigmentary retinitis caused or precipitated by trauma. In looking over the original work of Wagenmann, one can hardly agree that his interesting experiments have any bearing on the pathogenesis of pigmentary retinitis. Without going into detail it will be enough

10. Wagenmann, A.: Arch. f. Ophth. **36**:1, 1891.
11. Hine, Montague L.: Proc. Roy. Soc. Med. (Sect. Ophth.) **17**:14, 1924.

Fig. 3.—Fundus of the left eye in December, 1930.

TABLE 2.—*Summary of Observations in Cases of Unilateral Pigmentary Retinitis*

Case	Adaptation	Fundus	Ocular Complications	Pathologic Conditions Other Than Ocular	Period of Observation, Course	Comment
1	Impaired; not measured	Papilla showed pale yellow discoloration; pigment in the form of bone corpuscles throughout fundus, except in papilla and macula; retinal arteries about half the size of normal	.....	Deafness of both ears	.....	Through a coincidence this patient, first seen by Pedraglia in Rio de Janeiro in 1865, was found in 1890 by Deutschman in one of the hospitals of Hamburg; there the patient soon died; Deutschman examined his eyes pathologically
2	.....	Macula the only part of fundus free from pigmentation	Divergent strabismus; myopia, about 9.0 diopters	.....	.....	.....
3	.....	Papilla atrophic; arteries narrow; pigment in the form of bone corpuscles at the periphery of the retina	.....	Deafness of left ear	.....	.....
4	.....	Disk atrophic; a good deal of pigment in the equatorial zone	.....	.....	According to patient, brother had same condition, which had existed for many years and was becoming progressively worse	
5	Impaired; not measured	Disk waxy yellow; pigment in the form of bone corpuscles throughout the fundus, except in papilla and macula; vessels narrow	Posterior polar cataract; keratitis	Hemiplegia in the past	.....	.....
6	.....	Papilla of waxy appearance; arteries narrow; ramified pigment deposits occupying equatorial zone, particularly its inferior external sector	Convergent strabismus; hyperopic astigmatism	Pulmonary tuberculosis	.....	Patient died the same year; a pathologic examination revealed changes characteristic of pigmentary retinitis
7	.....	Belt of pigmentation complete; its anterior limit cannot be reached in ophthalmoscopic examination	Divergent squint, —2.0 diopters	.....	.....	.....
8	.....	Waxy disk; atrophic arteries; rather large masses of pigment	Posterior polar cataract	.....	.....	Injury over right frontal sinus in November, 1916; vision had failed since
9	Förster, right, 2 cm. opening	Papilla yellowish red; arteries narrow; pigmentation both in the form of bone corpuscles and in amorphous masses	Evidence of keratitis parenchymatosa in childhood; posterior polar cataract	.....	.....	.....
10	Förster, 22 mm.	Disk normal; arteries narrow; pigment throughout fundus except in papilla and macula	—2.0 diopters	.....	Ten years	.....
11	1/8 of normal limit	Waxy yellow disk; narrow arteries; pigment in the form of bone corpuscles	Posterior cortical cataract	.....	Six years; progressive limitation of visual fields	.....

to quote Wagenmann's own conclusions about the changes observed in rabbits' eyes following the severing of ciliary arteries: "Das Bild gleicht dem einer abgelaufener Chorioretinitis mit teilweisem Schwund des Pigments und klumpen Pigmenthäufchen." (The picture resembles that of a healed chorioretinitis with partial disappearance and lumpy accumulations of pigment.) This certainly does not resemble a true retinitis pigmentosa.

In the absence of any clinical or experimental support, the "traumatic" origin of pigmentary retinal degeneration appears to be no

TABLE 3.—*Cases Reported as Unilateral Pigmentary Retinitis and Not Included in Table 1*

Author	Year	Reason for Excluding Case from Group of Unilateral Pigmentary Retinitis
Pagenstecher (Klinische Beobachtungen, 1861, vol. 1, p. 53)	1861	Ophthalmoscopic picture atypical
Mooren (Ophthalmologische Beobachtungen, Berlin, A. Hirschwald, 1867, p. 261)		Not unilateral; visual field of "normal" eye constricted
Ancke (Centralbl. f. prakt. Augenh. **9**: 167, 1885)	1885	Atypical changes in fundus: white spots in equatorial zone
Neuffer (Ueber den Einfluss der Heredität und der Consanguinität der Eltern in der Aetiologie der Retinitis pigmentosa, Strasburg, 1893)	1893	Dense vitreous opacities; retinal separation
Reinecke (Retinitis pigmentosa, Kiel, 1894)	1894	Report too brief and not convincing; no reason given for reduction of visual acuity of "normal" eye
Rosenbaum (Retinitis pigmentosa, Kiel, 1900)	1900	Report too brief
Reuter (Arch. f. Augenh. **60**: 59, 1908)	1908	Affected eye had evidence of uveal involvement: posterior synechiae; clinical course of the "pigmentary retinitis" atypical: complete blindness in two years
Bakker (Tijdschr. nederl.-ind. **65**: 593, 1925)	1925	Not strictly unilateral: adaption of normal eye impaired

more than a misunderstanding. Thus, neither syphilis nor trauma can serve the purpose of excluding unilateral pigmentary retinitis from the group of genuine ocular degenerations. It remains to be seen how just is the assertion of Shoemaker and others that in cases of unilateral pigmentary retinitis the normal eye may eventually become affected.

It was brought out long ago by Höring,[12] and later confirmed by numerous investigators (Mooren,[13] Schneider,[14] Ernroth,[15] Leber,

12. Höring: Retinitis pigmentosa, Zehenders Monatsblätter **2**:233, 1864.

13. Mooren: Ophthalmologische Beobachtungen, Berlin, A. Hirschwald, 1867, p. 261.

14. Schneider: Ueber die Erblichkeit der Retinitis pigmentosa, Berlin, C. Vogt, 1896.

15. Ernroth: A Case of Pigmentary Retinitis in a Child Aged Four Years, Vestnik Oftal. **16**:308, 1899.

Nettleship[16] and others), that the first signs of pigmentary retinitis are observed at the age of 3 to 8 years. In a few cases, patients affected with pigmentary retinitis asserted that they were unaware of any visual defect up to middle or even advanced age. According to Nettleship, these were cases with a long and exceedingly slow course. In the large material on pigmentary retinitis collected by a generation of ophthalmologists, the onset of genuine pigmentary retinitis in an adult was not indisputably established. From table 1, however, it is evident that practically all cases of unilateral pigmentary retinitis were observed in adults. It is not surprising, under these circumstances, that whenever the opportunity of prolonged observation presented itself, the unilaterality of the retinal lesion could be definitely confirmed. Such was the case of Shoji in which the author examined his patient with a unilateral pigmentary retinitis after an interval of ten years and found no changes, either ophthalmoscopic or functional, in the normal eye. In my case the patient has been repeatedly examined for six years, and while the condition of the diseased eye is definitely progressing, no evidence of a similar involvement can be detected in the normal eye.

Thus it seems to be definitely established that in rare instances pigmentary retinitis, or at least a condition indistinguishable from it, affects only one eye. The importance of this fact lies in its relation to the unsolved problem of the pathogenesis of pigmentary retinitis. If the latter is to be placed without reservation among ophthalmic abiotrophies (Collins[17]), it is hard to understand how an inherent deficiency of "the vital force" can be limited to the neuro-epithelium of one eye. In fact, the unilateral occurrence of ocular diseases which belong unquestionably to the group of abiotrophies (for instance, Tay-Sachs' disease) is entirely unknown. I do not think that the important mass of evidence collected by Collins in favor of the abiotrophic nature of pigmentary retinitis is entirely without value, but it is not beyond a possibility that the clinical entity of pigmentary retinitis covers more than one pathogenic conception.

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16. Nettleship, E.: On Retinitis Pigmentosa and Allied Diseases, Roy. London Ophth. Hosp. Rep. **17**:1, 1907-1908.

17. Collins, E. Treacher: Hereditary Ocular Degenerations, Trans. International Congress of Ophthalmology, Washington, D. C., 1922, p. 103.

Ophthalmologic Review

DIAGNOSTIC METHODS *

JOHN N. EVANS, M.D.
BROOKLYN

In an excellently worded paper in the *Lancet,* Duke-Elder [1] showed that further progress in ophthalmology cannot be built on a groundwork of pathology. Fundamental knowledge and basic methods of study are now sufficiently secure so that one can look into the murkiness of pathologic states from the heights of secure data culled from the body in healthy activity. It is in the realm of physiologic chemistry that efforts will be most fruitful.

Evidence of disturbed physicochemic balance is becoming more and more available through forms of clinical study. The reawakened interest in light sense studies,[2] in the chemical composition of the aqueous, lens and vitreous,[3] in fluctuations of ocular tension [4] and in a great variety of refined methods of diagnosis [5] is strongly indicative of this trend.

It would be valuable if the various diagnostic methods could be reviewed here from this angle, for many of the complexities of everyday experiences would appear in a more wholesome light. I shall endeavor to consider only certain high spots in the field of diagnosis that seem to lead to better channels of thought and to more useful highways for progress.

VISUAL FIELDS

Passing back and forth along the arc of progress, perimetry and scotometry have been lost again and again in the very controversial shadows that they would define, but quite as often have emerged in the all-embracing expanse of proved truths. I shall review certain of these truths, not in their chronological order, but in their relation to the evolution of the subject.

Studies of the visual fields became a real aid in diagnosis only within the past ten to twenty years. Studies had been made for diagnostic purposes before that time, but they contributed little to the ultimate understanding of a given situation. The earliest investigators understood that the usefulness of these studies depended, first, on related anatomic and physiologic data. This was true not only for interpretation, but also for the application of a proper technic. Thus basic

* Submitted for publication, March 4, 1931.

studies had to be supplemented by numerous workers [6] in order to unfold the ultimate course and relations of the optic pathway, the arrangement of fiber bundles in the nerves, tracts and radiations and the projection of retinal zones at various levels. Injuries during the World War were of such a type as to further the studies on cortical representation of the retina. The same studies were carried on by workers [7] in various countries during the same years. Their conclusions matched satisfactorily.

As a better understanding of the fiber bundles in the optic pathway developed, a reawakening of interest appeared in the arrangement of fibers in the retina; [8] but in spite of the ease with which fibers could be seen with red-free light,[9] little more was learned than ophthalmologists had known fifty years earlier.[10] The difficulty did not seem to be in understanding the arrangement of the retinal nerve fibers, but rather in the topography of their terminations. What is the relation of a special retinal region to a particular fiber bundle? Do fibers split and send off branches? Do certain bundles overshoot other bundles? Is the retina supplied in concentric zones, in sectors or in bandlike swathes? In spite of noteworthy efforts to answer these questions, useful data were not supplied. Dependable evidence on the arrangement of fiber bundles in the optic pathway soon permitted the development of quantitative perimetry.[11] This was the greatest advance since the days of von Graefe,[12] for it permitted the localization of lesions of the brain in a way never before achieved. Certain defects soon became easy to interpret as being due to pressure on this or that fiber bundle, and soon the words "pressure on fiber bundles" became the open sesame to all defects of the fields. Such a wholesale acceptance of the slogan could only portend confusion. Defects originating in localized disturbances in the retina [13] were explained as due to "pressure on its fiber bundles."

It was taught that the most sensitive portion of a nerve link was the synapse. The common lesions of the brain which produced the classic defects of the fields were, for the most part, far removed from the synapses of the optic pathway, but in the retina the disturbance was intimately associated with these vital inosculations. Nevertheless, this significant fact was overlooked in the great enthusiasm for the slogan of "pressure on fiber bundles." It was during this stage that the "classic" defects of glaucoma were evolved [14] and though the very ophthalmologists who discovered them showed dissatisfaction with such interpretation and though it soon appeared that the same defects occurred in conditions in which there was no pressure,[15] the hue and cry of "pressure on fiber bundles" went on.

With such strong emphasis on but one factor, methods of study and the very technic began to suffer. In spite of the fact that the tangent screen was advocated for such studies, the perimeter became the popular instrument; its mechanical features were elaborated and exaggerated until the tool became the master of the worker. A series of bizarre geometric patterns—"visus reticulatus" [16] or the cribriform field—were presented as typical defects, and weird and baseless theories were offered to explain their genesis. Attention was occasionally called to the apparent relation of certain defects to retinal vessels [17] and to vascular disease; [18] but, as the bare statement that retinal function was disturbed in a circumscribed region did not satisfy the school that considered the fiber bundles responsible, little consideration was given to the vascular theories.[19]

The neuro-anatomists [20] and neurophysiologists [21] had worked out a satisfactory theory based on reliable data which showed an intimate relation between the cerebral vessels, perivascular spaces, cell spaces and synapse function. Though suspicion [22] had been aroused, it was much later [23] that the ophthalmologist presented data showing the complete analogy of these cerebral relations to those existing in the retina. Thus data were finally obtained that showed that the retinal perivascular-pericellular system was susceptible to influences that produce a suspension of synaptic function, with the resulting loss of retinal function and the formation of a corresponding scotoma. Sound stepping stones of data had been securely placed for a new advance.

TECHNIC

The extent and shape of the visual field had probably first been demonstrated as a physiologic curiosity. Its normal extent under various conditions was established and its limitations by the encroachment of nonperceptive structures were recognized as producing normal defects.* Deviations from the normal defects were interpreted as pathologic, and the clinical usefulness of these data was soon recognized. Acceptance of this basic law—recognition of deviations from the normal—permitted the evolution of quantitative perimetry and its successful application as soon as the necessary anatomic and physiologic data had been gained.

The earlier workers had also established the presence of another normal defect, the blindspot of Mariotte.[25] Variations in this normal scotoma soon proved to be of utmost value in clinical study.[26] The

* These defects or limits were due to the brow, nose, cheek, pupil and functionless retina (far temporally). Von Graefe was the first to bring out this point (Arch. f. Ophth. **2**:258, 1856).

same early workers, being unbiased by popular slogans, were less hampered in their observations. Helmholtz had outlined heavy shadows of vessels, [17c] and expressed no surprise that he should be able to do so. Tradition soon benumbed further attempts to plot shadows of vessels by the implied premise that, if retinal vessels were shadow-forming, their presence would obstruct vision. Various workers had succeeded in obtaining maps of the vessels,[17c] but their interest lagged when technical difficulties arose. Their clinical usefulness could not be recognized until detailed study had disclosed certain significant phenomena.[27]

DIRECTION OF MOVEMENT

It is significant that at an early stage workers had emphasized the necessity of moving the object at right angles to the defect.[12]

The satisfactory progress made with the perimeter was probably due to the incidental development of the device into an arc, which forced the movement of the object radially and thus at right angles to the peripheral normal defects which limit the field. The normal blindspot, being more or less oval, also encouraged the movement of the object at right angles to its borders (radially [17c] toward its center). It thus became apparent that the greatest progress—quantitative perimetry and studies of the blindspot—came about in those instances in which ophthalmologists had been forced into the necessary principle of moving the object at right angles to the borders of defects. In like manner, the same agencies had militated against the use of this principle in mapping irregular scotomas. The retinal vascular tree and its associated scotomas had in this way become obscured through lack of application of the most productive technic. Thus, it finally became obvious that one could map not only these angioscotomas but also other minute defects through application of the law of right angle mapping. To map shadows of vessels and the associated lesions demanded a familiarity with the three types of distribution of the retinal vessels.[27] One must visualize the normal defect while plotting variations called pathologic.

It gradually became evident that of equal importance with the law of right angle mapping was the law of size of the object.[28] It seemed obvious that if one were to map a certain defect which reduced vision to 20/40, one should not use an object that would correspond in size to 20/200, for it would be so large that the defect would be covered by the object and so would not be discovered. If the object corresponded in size to 20/70, it would perhaps become partly invisible or dim in passing the defect, so that one would record a relative scotoma. If, however, one used an object corresponding in size to 20/30 or 20/20, it would be entirely concealed by the 20/40 defect, so that

the subject would report its disappearance. The defect would then be absolute or positive—a much more reliable measure of its size and shape, with the elimination of an indefinite judgment of quality. The result would then have a quantitative value. With this general principle in mind, effort had been made to state the size of the object in minutes or degrees and to work out the zones of acuity at various distances from the macula.[28a] Besides the fact that the angular size of an object did not indicate its visual but only its geometric size[29] (possibly its optical size), one had to change its dimensions continually as one passed to different portions of the retina to understand the results. It was better to state its size in millimeters (the conditions of the test) and adopt the law of object size: use the smallest object that the patient can report on accurately.[30]

For convenience in comparing pathologic with normal conditions, the isopter zones of peripheral acuity were adopted.*

Hand in hand with the size of the object goes the fixation distance. There was some dispute about this. Short distances, from eye to campimeter, were admittedly the best for accuracy of fixation. Short distances, however, necessitated the use of minute objects. Long distances gave greater magnification of the defect but less steady fixation. It is only within the past few years that sufficiently minute objects have been available[27] for fixation at short distances. The difficulties of accommodation at short distances were overcome by the proper correcting lens.

Perhaps it was the sentimental side of man that caused so many workers to adhere to the mapping of color fields in spite of the fact that no one had presented evidence that allowed a logical interpretation of the color defect when found. Finally, a substantial advance in methods of studying the visual field resulted from the acceptance of certain truisms demonstrated by a number of technical and clinical studies, so that one was forced to make the following conclusions.

The use of colored test objects had little value as a clinical test, because:

(*a*) Their use involved complicated conditions that were not reproducible in subsequent examinations.[31]

(*b*) The necessary technic was too elaborate.

(*c*) Physiologic color fields varied so as to be indistinguishable from pathologic color fields, particularly in borderline cases.[31]

(*d*) Sufficient satisfactory clinical data was not available for the interpretation of defects in the color fields.[32]

(*e*) Any defect for color was present for white if a sufficiently small object was used.[33]

* The size of object to be selected in a particular study not only varies with the general region of the section mapped and with the condition of the eye under examination, but also with the mental alertness of the patient.

From similar material, other conclusions were permitted:

Peripheral field:

1. Uniform contraction of the fields was of relatively little significance for interpretation.[34]
2. The shape of the field was most significant:
   (*a*) The quantitative method of study not only was the most practical, but showed variations sufficiently early and definitely to be of value.
   (*b*) The anatomic and physiologic basis for the interpretation of defects of shape rested on a basis of secure evidence.[35]
   (*c*) The clinical value of quantitative perimetry had been satisfactorily established.[36]

Central field:

1. Defects of the central field had their origin in two separate mechanisms:
   (*a*) Defects of the fiber bundles,[37] and
   (b) Defects arising from local disturbances of the blood and "lymph" flow.[38]
2. Minute objects were to be advocated:
   (*a*) Not based on the visual angle.
   (*b*) Based on the fractional relation (in millimeters) of object to fixation distance.[39]
   (*c*) Any relative scotoma could be recognized as an absolute scotoma if sufficiently small objects were used; by this method the result was more reliable and more easily interpreted. (Thus the size of the object should not be written 1.5 degrees, but 3 mm./190 mm. The size in degrees gives the geometric relation and is probably not applicable; the fraction is a direct statement of the conditions under which the test is made.[40] The commoner proportions may range from 6 mm./330 mm. to 2 mm./2,000 mm.[41]
3. The classic sickle scotoma of glaucoma arising from the blindspot was usable as an index of the progress of the disease.[42] It was not pathognomonic of this condition alone, but of a vascular process [25] or, as lately suggested, a manifestation of disturbance of the perivascular space.[43]

INSTRUMENTS

It appeared that the reputation for lengthy and tedious examinations required for studies of the fields was mainly responsible for their unpopularity as a routine diagnostic method. When modern instruments and technic were used it soon seemed justifiable to state that quantitative studies of the fields need take no longer than six or seven minutes. This meant mapping with three sizes of white objects and recording the result for filing. Satisfactory and reliable plotting of the blindspot, ready for filing, took no longer than three or four minutes for each eye. If more time was consumed, it was felt that the technic was at fault or that one was not taking advantage of modern facilities.

Of the various instruments used in the study of visual fields, the perimeter has been most commonly employed, not because it was most

useful, but because it was available at various prices and numerous models were on the market. Besides this, the technic was apparently so simple as not to appear forbidding to the novice. The Rand-Ferree perimeter, without doubt, was the most nearly perfect single device available for collecting the data on normal and pathologic peripheral fields.

In it were assembled the developments of the past three quarters of a century. It was devised primarily for a technical type of work, and for that reason was frowned on by the clinicians. This attitude was distinctly unfair. It was not the complicated device which it appeared to be to those unfamiliar with its uses. It embodied a number of instruments.

Any of the tests and especially those of a functional kind must be carefully standardized so that the conditions will be the same at successive examinations. The Rand-Ferree perimeter made it possible to repeat an examination under the same conditions whenever necessary. The mechanical and optical arrangements used were simple and adequate. Means were provided for centering the patient's eye and maintaining fixation, for illuminating the arc and for altering the various relations to meet a wide variety of normal and pathologic conditions. The fixation could be arranged so as to meet the needs of persons with a high degree of myopia, with presbyopia or with a central scotoma. Beside these and numerous other features, means were provided for studies of the central field. While the advised methods of using this instrument were not universally accepted for clinical studies, the device itself was a worthwhile addition to the technic, not the least virtue of which was the elimination of mechanical devices for recording the data. It must not be inferred, however, that the Rand-Ferree instrument meets all the requirements for a perfect clinical instrument. Certain minor changes could be made to facilitate its routine use in the office.*

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*A clicking spring, based on the principle of the "click spring" on the ophthalmoscope wheel, is helpful in turning to the meridian to be followed (for clinical studies it would seem that mapping along twelve meridians has become the standard). Inconspicuous numbers along the inner surface of the arc also save time in taking the readings. By removing the screw on the side for varying the adjustment of the head rest, one can slide that support to shift rapidly from one eye to the other. If a prism of the proper strength is placed in the axial eyepiece (used to center the subject's eye) so that its edge bisects the opening, a double image of the patient's cornea is seen. When the double images just touch, the proper fixation is attained. It is possible to devise a small projection lamp that will throw a disk-shaped spot of light on the arc. This is used as an object, the size and intensity of which can be varied with proper diaphragms and filters.

The fixation distance of 330 mm. which this perimeter offers allows it to be used for refined studies, objects in graduated sizes of 1, 3 and 6 mm. being satisfactory and allowing sufficiently wide separation of the normal isopters for accurate studies of variation in pathologic conditions.

In using the perimeter, one can greatly speed up the process by developing a definite order of procedure. One should always begin with the same meridian and test the others in sequence, keeping the readings in mind and recording them when all have been taken so as not to interrupt the study to make notations.

In like manner the Lloyd stereocampimeter made it possible to standardize the technic of study of the central field. It provided many facilities for meeting those ever-changing conditions of clinical work. The resurrection [44] of the direct plotting method suggested by the early workers [45] made it unnecessary to copy the map. In this way an uninteresting and time-consuming element was eliminated.

It was unfortunate that both these instruments were locked up by patents so that their cost was disproportionately high. This has definitely retarded the more universal interest in studies of the fields. It is evident, however, that many of their good features can readily be applied to devices already available.

THE OPHTHALMOSCOPE AND METHODS OF OPHTHALMOSCOPY

Within ten years after Helmholtz had devised the ophthalmoscope,[46] a deluge of improvements had appeared. Elaborate table models, binocular arrangements for perception of depth, appliances for examining one's own eyegrounds and a flood of hand models represented the efforts of the pioneers to refine and to improve the instrument.[47] A sort of hiatus in its development then appeared.

Shortly before the World War a reawakening of interest was evident.[48] It was realized that a more satisfactory means of perceiving and measuring relief was needed, so that depressions and elevations could be studied minutely. Five basic means of observing relief had been worked out for various forms of examination: parallax, difference of focus, binocular observation, distortion of an optical section (geometrically shaped pencils) and shadows cast by oblique rays.

It is perhaps conceivable that one might refine methods of determining level by parallactic displacement. Later workers, however, did not give it the attention that earlier generations had. The advent of the slit-lamp was doubtless responsible for this, as the method was most used for studies of the anterior segment before biomicroscopy provided better means. Nevertheless, parallax deserved consideration and could doubtless be improved by further refinements.

Measuring by difference in focus of succeeding lenses had been considered definitely unsatisfactory for those whose accommodation was very active. Observers could not be sure of the data obtained by focusing the lens on the hazy surface of a swollen nerve head as compared to a more or less edematous region of the adjacent retina. Ophthalmologists were also taught to use the large table ophthalmoscope for binocular observations to aid in the differentiation of depth.* The latter method, while convincing, did not permit one to set down data for future comparison.

Dissatisfaction with "interpretation of level" evidently stimulated a number of workers to improve the methods. Perhaps first I should mention those who developed ophthalmoscopic photography. Of course this means stereophotography of the fundus, for by that method not only was direct evidence of elevation and depression secured, but a permanent record was obtained for comparison with later studies.[49] Measurements could be taken and changes recorded. An apparatus for stereoscopic photography with red-free light, with both focal and diffuse forms of illumination, was presented. It included the great advantage of a bulb with a metallic filament in place of the arc lamp. This combination obviously made a far more practical device for clinical work.[50]

Demonstration of alteration in depth had also been made possible by application of the same principles used in the slit-lamp: A sharply defined beam could be focused on a particular retinal or choroidal lesion so that details of structure and form could be sharply defined by distortion of the optical section. Elaboration of this method had already led to more refined diagnosis, just as the slit-lamp had improved understanding of disturbances of the media. A slit-lamp ophthalmoscope [51] of the hand type accomplished this end. It also created the conditions of oblique illumination. It was a real advance and should receive popular acceptance. With this principle in mind, a writer [52] in 1920 suggested the slit-lamp beam as a source of light for ophthalmoscopy with the mirror.

The polyophthalmoscope [53] allowed many observers to study a detail at the same instant, and not only permitted a more intelligent consultation, but provided a practical means of instruction for small groups of students. While these devices were more or less elaborate, they

* It is surprising that with the advent of the electric hand ophthalmoscope there has not been a revival of the apparently satisfactory binocular ophthalmoscopes that were used with mirrors. The one devised by Giraud-Teulon, and that of Loarence and Heirsch were evidently popular at one time (Helmholtz, H.: Treatise on Physiological Optics, p. 249. Wells, S.: Treatise on the Diseases of the Eye, p. 300).

were definitely practical and allowed the actual measurement and plotting of changes. The often suggested expedient of projecting a scale on a region of the fundus was made more practical by these instruments.[48a] It will be recalled that in 1920 [54] a writer suggested that one etch (or lay on with ink) 3 mm. squares on the object lens used for indirect ophthalmoscopy and thereby project a grid on the eyegrounds. A similar device—a disk with a minute grid etched on it—placed on or above the condensing lens of the electric ophthalmoscope was also advocated to accomplish the same result.[55] Both of these were improvements on an older method which used a secondary mirror to project an offset scale.[56] While none of these methods became universally popular, their helpfulness under certain conditions could not be denied.

During the heyday of the reflecting ophthalmoscope, energy was directed to refinements of instrument and technic. The electrically illuminated hand ophthalmoscope intensified the difficulty of annoying corneal reflexes, so that a concerted effort developed to avoid this consequence. The outcome was the development of large and small reflexless models. Stereoscopic ophthalmoscopy and accurate measurements of area and depth became practical.[48a] The popularity of these instruments only waited for forms requiring less elaborate adjustment.

The use of light of limited wavelength was empiric until Vogt,[57] in 1913, advocated the use of red-free light. An intense light became important for this form of study. The temperature of the source had to be at least from 3,000 to 4,000 C. or insufficient light would pass the filter, and minute details would not be visible. The cumbersome and expensive arc lamp proved to be the most practical light, except in certain localities in which direct sunlight was available. A simple system of mirrors and shutters conducted the sunlight into the examining room.[58] An adaptation of a very small arc lamp [59] was suggested, and numerous attempts [60] were made to use the more powerful forms of filament lamps. For the latter source, the bulbs of the moving picture projector (3,000 watts or more) were used.[58b] One of the most convenient seemed to be the slit-lamp bulb.* The temperature of its filament reaches 3,050 C. (Zeiss), so that nearly 80 per cent of its light passes the filter (blue-green). In order to use this bulb, special provision had to be made. The light was slipped into a tube and a plus 15 diopter lens was set 7 cm. in front of it.[58b] The filter was then

* These bulbs are inexpensive and fit the standard lamp socket. It is necessary to reduce the house current. The rheostats as supplied are not very expensive, but a satisfactory one can be made from the heating element of the electric flatiron. A few trials will demonstrate the number of coils that will give the best results when connected in series.

inserted. One used the reflecting hand ophthalmoscope with this device by the direct method. The source, mirror and subject were placed close together so that little light was lost.

The mercury vapor lamp was used [61] even before the arc lamp was popular. It had the advantage of needing no filters, but was expensive and cumbersome to manage. The large simplified Gullstrand ophthalmoscope was modified so that it could be used in conjunction with an arc lamp and the proper filters for studies with red-free light.[62]

The slit-lamp ophthalmoscope referred to was also adapted for examination [51] with red-free light.

Attempts to use the miniature filament lamps of the hand electric ophthalmoscope gave unsatisfactory results. The filters reduced the illumination so greatly that the fundus was seen in red-poor but not in red-free light, and nerve fibers and other details were not brought out sufficiently for one to study them.

Two general types of red-free filters have been advocated: the blue-green, the first one developed, and the yellow-green. The following are in use:

1. Blue-green: [63]
   (*a*) An aqueous solution of the dye erioviridin 1 per cent in a glass cell (1 cm. in depth).
   (*b*) An aqueous solution of copper sulphate 30 per cent in a glass cell (1 cm. in depth, used with *a*).
   (*c*) This combination (*a* and *b*) filter was also made up as a gelatin layer colored with the dye and placed between two pieces of heat-absorbing glass (Zeiss) (this glass takes the place of the copper solution).
2. Yellow-green: [59]
   (*a*) A glycerin solution of the dye aniline green naphthal B.* (This is an intense dye.) A minute quantity is to be mixed with glycerin and this solution added to glycerin very slowly until a red 2 cent stamp looks black when viewed through it by the light source to be used. Another suggestion is that a red geranium should look black through it in sunlight.[64]
   (*b*) Wratten filter 52 (this is satisfactory when combined with a filament light source); [59] Wratten filter 22 (see Friedenwald [65] for other filters).
   (*c*) Kodak Wratten celluloid filter 61.[64]

The copper solution was used primarily to absorb the heat (infrared) but also removed some of the visible red. Glass containers for the solution or glass screens had to be placed somewhere in the system in order to remove the dangerous ultraviolet.†

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* This is manufactured by Eimer and Amend, New York.

† The focusing lenses and solution containers provided with irradiation lamps are made of quartz and must be replaced with glass when used for study of the fundus.

Assuming, then, a satisfactory source burning at a temperature at or above 3,000 C., any of these filters would bring out details ordinarily unobserved in the red-orange light with which the fundus was flooded by the ordinary methods.

When red filters [65]—Wratten 29F—were used, the vessels and minute hemorrhages were subdued, but pigment was exaggerated. These were therefore found useful for a study of pigmentary changes at the macula. With a red-free filter, however, the opposite condition prevailed; the hemorrhages and vessels appeared black, and the pigment subdued.

Blue-green light brought out the yellow color of the macula best, and also exaggerated the nerve fibers.

Yellow-green light emphasized capillaries and hemorrhages, and exaggerated retinal membranes, exudates and detachments.*

The basic evidence on which the clinical diagnosis of pathologic conditions could be made may be summed up as follows:

The macula:

A. Normal:
1. Uniform disklike area of yellow pigmentation
2. Point reflex surrounded by
3. Double halo reflex

B. Pathologic:
1. Scattering and grainlike appearance of yellow pigmented area
2. Absence of point and halo reflexes
3. Rosette-shaped folds about the macula in
   (*a*) Acute condition of retina and choroid, and in
   (*b*) Acute condition of anterior parts of eye

Nerve fibers:

A. Normal: distinct radiating white lines

B. Pathologic:
1. Absence or decrease of visibility of fibers
   (*a*) Localized; absence of: multiple sclerosis, retinal edema
   (*b*) General; absence of optic atrophy
   (*c*) Absence restricted to significant area; hemianopia of infranuclear lesions [9]
   (*d*) Absence of papillomacular bundle; retrobulbar neuritis
2. Increase of visibility of fibers; early acute inflammation

Vessels:

A. Normal: appear black; capillaries visible [66]

B. Pathologic: exaggeration of details in sclerosis, hemorrhage and newly formed vessels

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* One is tempted to predict that a two-color filter (such as cobalt glass) might enhance the effect of relief by exaggeration of parallax, contrast, and possibly under certain conditions two points at different levels might be held in focus at the same time, as one red and one blue.

Stated more broadly then, it became obvious that the demonstration of exudates helped to differentiate inflammatory processes from simple congestive changes, such as papillitis from papilledema. Disappearance of fiber reflexes from the corresponding area of the retina helped to localize lesions above or below the primary centers.[9] Choroidal pigment was said to be rendered less visible and retinal pigment more visible.[63]

Certain difficulties seemed to militate against the more general use of light of limited wavelength for ophthalmoscopy. The source of light was, for the most part, cumbersome and unwieldy (arc lights). It was also inconvenient to manipulate because of the high temperature that it attained. The intensity of the light used was so great as to cause the patient discomfort, not only through its dazzling effect, but because it left a strong after-image. Moreover, so much of the entering light was absorbed that one found great variation in the distinctness of details in different persons owing to variations of pigmentation. The fact that the light was emitted from the source as a relatively narrow beam added difficulty which required considerable practice to overcome on the part of the younger ophthalmologist who had not been adequately trained in the use of the reflecting mirror. It had to be admitted that none of these objections was valid when such valuable evidence was available.

The diagnosis of pathologic conditions of the eyegrounds from changes in tint and shades of color was extremely unreliable, but two suggestions came forth to place this on a more secure basis: 1. A small attachment for the hand ophthalmoscope [67] converted that instrument into a colorimeter. An estimate of the relative degree of pallor of the optic nerve could be recorded and compared with a similar observation taken at a later date. 2. A more accurate and direct colorimetric measurement was made possible by use of the slit-lamp ophthalmoscope and appropriate standards.[48] A series of observations with this instrument might explain the apparent lack of relation between the appearance of an atrophied nerve head and the visual acuity. It might also permit a more satisfactory classification of optic atrophy and help in predicting the course of glaucoma.

While color photography of the fundus is yet in its infancy,[68] certain promising studies have been made. The main difficulty seems to be underexposure. This defect could probably be best corrected by the use of the new ultrasensitive photographic films by the use of some of the newer flashlight bulbs or by admitting light to the eyegrounds by transillumination of the sclera,[69] and by adapting the pinhole camera.

Certain minor refinements were suggested for the hand ophthalmoscope, which enhanced its value. An accessory prism of 20 degrees, held with its base in the direction of the object sought, permitted direct

examination of more peripheral retina than had heretofore been possible; this was a particularly opportune suggestion in view of the interest in locating retinal tears in cases of detachment. One needs every encouragement in this respect, as many hours may be spent before the opening is located. The same writer [70] suggested that a snap-catch be provided for the position of the lens-wheel which indicates when the aperture is before the eye. This enabled one to find the primary position in the dark of the examining room.

For a number of years, French writers [71] had advocated an accessory, the ophthalmodynamometer, to be used with the ophthalmoscope. One could make a measured amount of pressure with this tonometer-like device on the eyeball while focusing on a retinal vessel. The degree of pressure necessary to cause the pulsation of the vessel to appear and disappear was noted. These investigators demonstrated that normally diastolic pressure in an artery was greater than the intra-ocular tension, and that they became equalized if vascular pressure dropped in syncope and cardiac disease.[72] From studies on animals it was possible to devise a scale of equivalents so that the readings of the dynamometer could be translated into terms of blood pressure of the retinal artery on the disk. It thus became evident that this accessory to the ophthalmoscope could be of aid in the diagnosis of hypertension of the retinal vessels, particularly in the presence of systemic hypertension; but it was not demonstrated that retinal hypertension could occur unaccompanied by systemic hypertension.[73] This pulsation phenomenon occurred in optic neuritis with increased intracranial pressure.[74] The relation of variations in the pressure of the retinal blood vessels to changes in intra-ocular pressure was also made obvious.

One is apt to forget that the fundus may be examined by devices other than the classic ophthalmoscope. While such measures are useful only in emergencies, they should also be considered as offering possible means of obtaining evidence under certain conditions wherever the ophthalmoscope is restricted on account of its physical limitations. On one occasion, I obtained a satisfactory view of the fundus by using a stack of glass plates (microscopic slides) as a mirror after the manner of the original Helmholtz ophthalmoscope. The lack of disturbing reflexes and the uniformity of illumination were pleasing. On another occasion, the electric otoscope proved satisfactory for study of the fundus in a bedridden patient. The tip of a transilluminator light was recommended by one worker who applied it to the globe over the point to be studied.[75] When the subject was blonde, the examiner could get a satisfactory view of the fundus by looking directly into the pupil. This principle could be used to advantage in looking for a retinal tear far toward the periphery,[76] where this form of illumination is most

easily applied. It should be augmented by the use of a prism as already suggested. The Lang transilluminator could also be used for direct ophthalmoscopy if the examiner directed its curved point toward the patient's eye as he sighted over the tip.[77]

One is apt to look with scorn on the old-fashioned hydrophthalmoscope, but perhaps recent interest in the more peripheral portions of the retina and even in the ciliary body and angle of the anterior chamber may be a sufficient stimulus to cause reconsideration of the apparatus. The contact glass, as suggested for use in slit-lamp studies of the fundus and the angle of the anterior chamber, has practically been discarded because of the discomfort that it causes. The orthoscope [78] or hydrophthalmoscope, it will be recalled, was a glass box that was applied with its open face over the eye (as an eye cup would be). The box was then filled with a warm nonirritating solution through which the fundus could be studied. This device gave a clear view of the fundus in cases of a high degree of myopia and in cases of irregular astigmatism. It gave a view of the angle of the anterior chamber.[79] One can readily understand that such a device would have no more terrors for the patient than an ordinary eye bath, and that there would be little or no irritation, particularly if butyn or another modern anesthetic was instilled beforehand.

In order to facilitate localization of lesions discovered with the ophthalmoscope, the perimeter has been used.[80] Apparently, von Graefe [81] was the first to suggest this idea.

When a year has passed, each ophthalmologist should be able to look back and see that definite additions have been made to diagnostic methods. While this review is doubtless incomplete, it is hoped that the progress of ophthalmology as here outlined will make obvious the needs of the individual.

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News and Notes

EDITED BY DR. JOHN HERBERT WAITE

SOCIETY NEWS

Herman Knapp Memorial Alumni Association.—This society held its second annual meeting in New York on June 9, 1931. The afternoon was devoted to a clinical session at the hospital and there was a banquet in the evening. The officers for the ensuing year are: president, Dr. Walter S. Atkinson, Watertown, N. Y.; vice president, Dr. David F. Gillette, Syracuse, N. Y., and secretary-treasurer, Dr. William F. C. Steinbugler, New York.

Connecticut State Medical Society.—The annual meeting of the Eye, Ear, Nose and Throat Section of the Connecticut State Medical Society was held at the Stratfield Hotel, Bridgeport, May 21, 1931. After an interesting program, consisting of papers on hyperphoria, simple glaucoma and unilateral exophthalmos, the following elections took place: chairman, Dr. M. H. Merriman, Waterbury; vice chairman, Dr. A. N. Alling, New Haven; secretary and treasurer (reelected), Dr. Walter L. Hogan, Hartford.

Pacific Coast Oto-Ophthalmological Society.—Dr. Walter F. Hoffman, Seattle, was elected president at the meeting of the Society, May 28 to 30, in Los Angeles. The Society will meet in Seattle in 1932.

GENERAL NEWS

Dr. Fairchild Visits Indian Clinics.—Dr. Norah Fairchild, who is now in Vienna, visited during the winter months the Holland Clinic at Quetta, India, that of Dr. Mattera Dass in Punjab, and that of Lieutenant-Colonel Kirwan at Calcutta. At the mission hospital at Brindaban, Dr. Fairchild was invited to take charge of ophthalmic surgery during the coming winter. The clinics are not so well attended as in previous years, owing to economic conditions.

Graduate Course in Ophthalmology.—A graduate course in ophthalmology is to be given by members of the faculties of the four medical schools in Chicago, beginning September 1, to run for a year. Lectures and clinics will be held at the four universities and ten hospitals on a rotating schedule. Didactic instruction will be given from 8 to 9:30 a. m. and from 5 to 6:30 p. m.; clinical instruction will be given between 10 a. m. and noon and between 2 and 4 p. m. The course is limited to twelve students. The fee is $1,000, of which $100 is to be paid on application. Further information may be obtained from Dr. Richard C. Gamble, secretary, 30 North Michigan Avenue, Chicago.

Progress in Saving Sight.—Various avenues of approach to the problem of saving sight are discussed in the annual report of the National Society for the Prevention of Blindness. A study made during

the year by the Society in cooperation with the State and Provincial Health Authorities of North America indicated that fifty-three of the sixty-one states, territories and provinces require the use of a prophylactic in the eyes of babies at birth. Only five states and provinces have no laws or regulations making ophthalmia neonatorum reportable. Reporting is far from complete, however, the report points out. Demonstrations of eye testing for preschool children were conducted by the Society in seventy-one cities and ten states. Sixteen new sight-saving classes for school children have been added to the 350 already in operation in the United States. Four universities have given teacher-training courses for these classes. A special report on sight-saving classes was made at the White House Conference on Child Health and Protection. A study of conservation of vision in industry, involving the experience of 583 plants, was finished in 1930 in cooperation with the National Safety Council. The Society has also participated in research and demonstration projects on glaucoma and trachoma and has encouraged the development of social service in eye clinics. Public education through publications, exhibits, lectures, films and newspaper publicity has been extensive and is considered proof of growing interest in the work of the Society.

PERSONAL

Dr. John M. Wheeler, professor of ophthalmology in the Columbia University College of Physicians and Surgeons, was recently appointed to the board of directors of the National Society for the Prevention of Blindness.

CORRECTION

Two errors occurred in the article by Dr. Alexander Duane entitled "Binocular Vision and Projection" (ARCH. OPHTH. **5:** 734 [May] 1931). On page 748, footnote 6, at the bottom of the page, should refer to the September, instead of to the April, 1919, issue of the *American Journal of Ophthalmology*. On page 751, eighteenth line, the last word should be seconds instead of inches.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Aqueous Humor

THE PATHOGENESIS OF OPTIC ATROPHY IN OXYCEPHALY. M. J. NORDMANN, Rev. d'oto-neuro-opht. **8:** 1 (Jan.) 1930.

A report of this case was published previously; the present report is limited to the state of the eyes. The left globe projected more than the right, and both exhibited intense nystagmus. The anterior segments were normal, and the pupils reacted well to light and in accommodation. The papillae were a dirty white and somewhat irregular, and the caliber of the vessels was normal. In the periphery of the right eyeground, there was a spot of pigment. Vision in the right eye was reduced to movements of the hand at 20 cm., and in the left eye it was 5/5. In the left eye there was a concentric reduction of the visual field and a central scotoma for green and red.

The theories of the pathogenesis of optic atrophy in oxycephaly may be reduced to three hypotheses: compression of the nerve, infection and optic neuritis or intracranial hypertension and papillary stasis. The compression theory is based on the observation of narrowed optic foramina, the penetration of the optic canal by the carotid artery, dilatation of the third ventricle, enlargement of the hypophysis and traction on the optic nerve from deepening of the middle fossa. Compression of the nerve at the base of the cranium almost always produces simple, not gray, atrophy; optic atrophy in oxycephaly is usually postneuritic; simple atrophy is found in only from 10 to 15 per cent of the cases.

The question to solve is whether hypertension or infection produces the optic atrophy. Papillary stasis may accompany infectious and hypertensive processes, but it is, at least in the beginning, a noninflammatory edema. The conservation of good visual acuity while stasis exists and the noninfectious character of the intracranial hypertension suggest the absence of inflammatory elements in the optic nerve.

In a careful study of the cases reported by 150 writers, the author did not find certain signs of infection in any, although hypertension was frequently noted. This causal hypertension is due to disproportion between the growing brain and the rigid osseous skull. The author concludes: 1. The optic atrophy of oxycephaly is, in the great majority of cases, a gray atrophy. 2. Compression of the optic nerve usually causes simple atrophy and therefore does not explain the optic atrophy in oxycephaly. 3. The theory of isolated infection is not based on clinical fact but on a single questionable anatomic observation. 4. The optic atrophy probably is the consequence of a papillary stasis arising from intracranial hypertension, because (*a*) signs of hypertension are found constantly, (*b*) it has been demonstrated in some cases that this hypertension is aseptic and (*c*) cases of papillary stasis with conservation of good visual acuity certainly exist. 5. To solve this problem definitely, precise and complete clinical and pathologic observations are of the greatest importance.

DENNIS [Arch. Neurol.]

Bacteriology and Serology

VALUATION OF THE RAPIDITY OF THE PRECIPITATION OF THE RED BLOOD CORPUSCLES IN DISEASES OF THE EYE. G. PAVKOVITS-BUGARZKY, Klin. Monatsbl. f. Augenh. **85:** 528 (Oct.) 1930.

The rapidity with which red blood corpuscles precipitate has been recognized as valuable diagnostic and prognostic means in diverse branches of modern medical science. The opinion of modern writers still differs greatly as to the cause of this phenomenon. The author shows the development of the reaction. He mentions as its first observer, Hewson, who, in 1772, noticed what he called "crusta sanguinis phlogistica" in the blood taken by venesection from patients afflicted with certain, especially inflammatory, diseases. Considering the fact that only a few experiments have been made with this reaction in ophthalmology, the author undertook to examine the following questions: Is the rapidity of the precipitation of the red blood corpuscles influenced by diseases of the eye? Is its speed characteristic for certain ocular diseases? Can the reaction be used as a basis for differential diagnosis? Is there sufficient variation in different stages of the ocular disease that it may be used for a prognosis?

The precipitation was quickened in trachoma with ulcerating pannus, in lymphatic conjunctivitis and keratitis, in ocular diseases caused by syphilis, tuberculosis, rheumatism and nephritis and especially in parenchymatous keratitis, iritis and iridocyclitis. Normal conditions existed in catarrhal ophthalmia, xerosis, chemical burns of the conjunctiva and cornea and abrasion of the cornea; a moderate increase in rapidity was found in traumatic hemophthalmia, which became normal after the cure. The acceleration of the precipitation was most marked in sty, dacryocystitis, serpent ulcer and other purulent processes. It seemed to be present in all conditions connected with destruction of the tissues, whether the process was distinctly purulent or not; for instance, in parenchymatous keratitis.

The precipitation test is of differential diagnostic and prognostic value in rheumatic iritis, in which the rapidity does not diminish correspondingly with the decrease of the subjective and objective symptoms, indicating a recurrence. Decided acceleration, due to somatic symptoms, was found in three cases of cataract; slight acceleration in each case was followed by normal precipitation a month later. Increased acceleration after the operation was indicative of the development of iritis.

The prognostic value of this reaction was most evident in lymphatism, iritis, parenchymatous keratitis and other diseases with a tendency to recur. The result of the reaction indicated mostly either beginning recovery or approaching relapse. Similar information may be gained after operations. The author's experiments are based on the examination of more than 300 patients.

K. L. STOLL.

THE VALUE OF THE SEDIMENTATION TEST IN OPHTHALMOLOGY. N. POCHISSOFF, Russk. opht. j. **13:** 48 (Jan.) 1931.

In 1929, Franceschetti and Gugenheim reported the results of sedimentation tests with erythrocytes on 1,000 patients with diseases of the eyes. They expressed the opinion that this test should play an important rôle in the differential diagnosis of various ocular diseases.

The author studied 150 patients, 64, or 42.7 per cent, of whom had glaucoma; 43, or 29 per cent, cataract; 4, or 2.1 per cent, parenchymatous keratitis; 7, or 4.5 per cent, serpiginous ulcer; 2, or 1.3 per cent, malignant tumors; 23, or 17.3 per cent, various inflammatory diseases of the eyes, and 7, or 4.5 per cent, noninflammatory diseases.

The sedimentation test was performed on hospitalized patients in the morning by the micromethod of Panchenkoff. The technic of the method is described. Parallel tests were made for hemagglutinin and its influence on the sedimentation test, and the influence of postoperative complications on the test was determined.

The author draws the following conclusions: 1. The sedimentation test is not specific for glaucoma or for cataract. The acceleration of the sedimentation test in these cases was probably produced by a pathologic process elsewhere in the body. 2. This test is specific for inflammations and malignant neoplasms of the eye, because in 20 of 25 cases of an inflammatory character the sedimentation test was accelerated, particularly in parenchymatous keratitis and in serpiginous ulcers. In noninflammatory cases, the test was always normal. 3. Postoperative complications always give positive reactions to the sedimentation test. 4. Owing to a pathologic process elsewhere in the body the accelerated test does not influence postoperative healing of the eye. 5. The group of blood undoubtedly influences the test. 6. The sedimentation test should be included among other laboratory tests.

O. SITCHEVSKA.

Conjunctiva

AN ALGERIAN EPIDEMIC OF ACUTE FOLLICULAR CONJUNCTIVITIS. P. TOULANT, Arch. d'opht. **47:** 793 (Nov.) 1930.

Toulant observed eleven cases of acute follicular conjunctivitis between February and May, 1930; these were characterized by sudden onset, subconjunctival infiltration, ptosis with the appearance of follicles from four to eight days after the onset and with almost coincident swelling of the preauricular gland. Bacteriologic investigation gave negative results. These cases were similar to those described by Morax and Beal, but certain differences leave the author unable to decide this question. Swimming-pool conjunctivitis differs from it in the presence of inclusion bodies. There is also a resemblance to acute trachoma. The limbal lesions and the evolution are the deciding features in the diagnosis. Treatment by the application of 2 per cent silver nitrate and 2 per cent copper sulphate in glycerin was effective. The duration of the disease was at least two or three months.

S. B. MARLOW.

Cornea and Sclera

FURTHER OBSERVATIONS ON TATTOOING THE CORNEA WITH GOLD CHLORIDE: KNAPP'S METHOD. P. S. SOUDAKOFF, Nat. M. J., China **17:** 75 (Feb.) 1931.

Soudakoff, adding to his five cases previously reported (*Nat. M. J., China* **13:** 723, 1927), brings his total treated in this way to thirty. In reviewing the literature, he shrewdly observes that in most of the cases reported the patients were kept under observation for a short

time only, usually not exceeding six months. He followed up six of his own patients for as long as eighteen months, during which time four of them still had good cosmetic results. Nevertheless, he considers this too short a time to reach a definite conclusion concerning the efficacy of this method, and he quotes a case of Sallmann's in which the tattooed area had turned red at the end of three years. From his own experience, the author suggests that: 1. The solution of gold chloride should not be older than six weeks. 2. The solution of hydrazine hydrate should be as fresh as possible and should be kept in a bottle, the inside of which is coated with paraffin. 3. The epithelium should be scraped off under the control of the fluorescein. 4. The tattooed area should be marked with the help of von Hippel's trephine. 5. As much as possible of the corneal scar should be removed. He concludes that the chemical method, in its present status, has no advantage over the old mechanical method of tattooing.

G. M. BRUCE.

INTERSTITIAL KERATITIS IN HEREDITARY SYPHILIS WITH POSTERIOR ULCERS OF THE CORNEA. G. TIRELLI, Arch. di ottal. **38:** 38 (Jan.) 1931.

Only eight cases of this condition could be found in the literature. The author's patient was a girl of 15 showing other obvious signs of hereditary syphilis and a positive Wassermann reaction. Besides the ordinary findings of interstitial keratitis, the left cornea showed a central opacity in the deepest layers. Corresponding to this opacity a U-shaped rupture of Descemet's membrane could be seen with the slit-lamp, the vertical arms measuring 1 mm., and the horizontal connection 0.5 mm. Vision was limited to hand movements. Six months later a dense central opacity was still present, with a denser white line at the site of the rupture of Descemet's membrane. Vision was 1/20, gradually increasing to 1/5.

Such ruptures were seen by Igersheimer in his experimental animals with interstitial keratitis. They occurred at the site of most intense infiltration and were accompanied by clumping of cells on the posterior surface of the cornea. It seemed probable that such clumps of leukocytes were attracted to this site from the aqueous by the inflammatory changes, and that their digestive processes caused the defects in Descemet's membrane.

Elschnig believed that an increase of intra-ocular tension could be responsible for a process of necrosis in the cornea, which could cause such ruptures. The author's case showed an increase of tension to 30 in one eye, as compared to 22 in the other eye, which ran the typical course of the disease.

S. R. GIFFORD.

General Diseases

HYPERTENSION AND THE EYE: HYPERTENSION, HYPERTONIA AND THEIR ORGANIC SIGNS. J. PAL, Med. Klin. **27:** 193 (Feb. 6) 1931.

Pal differentiates between primary hypertonia and toxogenic hypertension. It has been found that not anatomic changes in the vascular system but rather functional disturbances of the arterial walls are the

primary cause of hypertonia. It is of course possible that gradually there will develop anatomic changes. The form of hypertension that develops as the result of functional disorders is generally designated as essential hypertension; however, the author prefers the term "primary" hypertonia. Many patients with this form of hypertension are constantly flushed; their faces, especially, are always red. Venous congestion does not exist in these patients, and the redness of the face is probably due to arterial stasis. Whereas in the primary form there exist mainly functional arterial disturbances in toxogenic hypertension, which is generally observed in renal disturbances and in lead poisoning, the entire cardiovascular system is involved, heart, arteries, capillaries and veins. The toxogenic cases vary in many points, especially in the behavior of the veins, from the cases of primary hypertonia. It has been found that characteristic changes in the fundus oculi permit a differentiation of the two types. In primary hypertonia the fundus of both eyes shows corkscrew-like windings in the distal portions of the smallest veins. In toxogenic hypertension, however, this sign is not detectable.

[J.A.M.A.]

OBSERVATIONS ON FUNDUS OCULI IN HYPERTENSION AND IN HYPERTONIA. G. GUIST, Med. Klin. **27**: 195 (Feb. 6) 1931.

Guist gives a more detailed description of the retinal vessels in patients with hypertension. He found that in primary hypertonia in which the walls of the veins are normal the fundus oculi reveals corkscrew-like windings in the distal portion of the small veins. The mechanism of this phenomenon is explained. In toxogenic hypertension in which the walls of both the arteries and the veins are thicker, the corkscrew-like windings are not to be found.

[J.A.M.A.]

Glaucoma

THE DEVELOPMENT OF THE EXCAVATION AND ATROPHY OF THE OPTIC NERVE IN SIMPLE GLAUCOMA. W. KAPUSCINSKI, Arch. d'opht. **47**: 779 (Nov.) 1930.

The histolytic properties attributed to the intra-ocular fluids by Elschnig are discussed by Kapuscinski in the first part of this paper. Experiments are cited which tend to disprove the presence of such properties. The author feels that all the anatomic changes in the optic nerve are due to the increased intra-ocular pressure. In support of his belief he gives the histologic observations in an eye that had had a hypertension of 52 mm. of mercury. The chief pathologic changes were in the disk where the nerve fibers had disappeared, especially in the center and in the cribriform layer. This layer had not bulged but had enlarged because of glial tissue, with compression of the nerve fibers; the nasal wall was drawn under the choroid. The nasal fibers last longest, perhaps owing to protection by the blood vessels. The author thinks that the atrophy of the nervous fibers is not related to their length. He points out that the physical laws of fluid under pressure demonstrate that the pressure is equal in all directions. For this reason the pressure on the nerve fibers cannot be greater on those resting on the scleral ring than on others.

S. B. MARLOW.

THE NONOPERATIVE TREATMENT OF GLAUCOMA. N. BLATT, Terapia 7: 384, 1930.

In treating patients with glaucoma, nonoperative general treatment should be considered along with the operative procedures that result in palliative effects, a decrease in tension and an improvement of acuity of vision, and yet are devoid of influence on the general cause of glaucoma of systemic origin. Nonoperative treatment includes local therapy in the form of instillations, for which pilocarpine and eserine are still predominant. In glaucomatous iritis and secondary glaucoma, the preparation glaucosan (an epinephrine substitute) recommended by Hamburger has stood its ground in practice. Its principle is epinephrine. Pituitary extract also decreases the tension of the glaucomatous eye, but it is not serviceable in practice because of the marked inflammatory reaction of the eyeball. In severe seizures of acute glaucoma, histamine (Hamburger) proves useful by engendering marked miosis and a decrease in tension, thus preparing a more favorable ground for operation.

To the nonoperative therapy of glaucoma belongs also phototherapy. It is used far too little, although in some cases it may narrow the pupil and decrease the tension by way of reflex and vasomotor conduction.

General treatment should go hand in hand with local and operative procedures, as it is already generally accepted that glaucoma is only a comprehensive term for a symptom complex of a still unknown systemic disease. The principle underlying this general treatment aims at the diminution of the intra-ocular liquid and a regulation of the current of this fluid. This aim can be attained by the incorporation of remedies by mouth or parenterally, and partly by diet, organotherapy or bloodletting. The diet should include much salt, little liquid and many vegetables. Gynergen tablets should be given internally; iodine is used only when arteriosclerosis is present. Bloodletting (from 200 to 300 cc.) should be done every six or eight weeks. If the tension remains high, operation should be performed. Cyclodialysis is the method of choice, although it is necessary to repeat this operation. Iridectomy is effective only in cases of acute glaucoma. In cases of secondary glaucoma one should try epinephrine, glaucosan and then operation. In cases of simple glaucoma, organotherapy should be added to local therapy, but operation should be shunned, if possible. Writing, reading and manual work should not be forbidden to any glaucomatous patient; the wearing of dark spectacles is not permissible (miosis through accommodation and light action). Exercise in the open and moderate sport are allowed. Women with glaucoma are not warned against pregnancy (hypotension in pregnancy). General treatment is an adjuvant to the therapy of glaucoma, but cannot replace antiglaucomatous operations.

N. BLATT.

Lacrimal Apparatus

RADIOGRAPHY OF PATHOLOGIC LACRIMAL PASSAGES INTO WHICH IODIZED OIL HAS BEEN INJECTED. M. DEL DUCA, Rev. oto-neuro-oft. 7: 421 (Sept.-Dec.) 1930.

The injection of "iodized poppy seed oil (40 per cent iodine)" has assumed great importance in the study of normal and pathologic conditions. In normal ducts the time taken to eliminate iodized oil is from

one-half to two minutes, according to the case. In a study of stenoses and ectasias, the author divides lacrimal sacs into two groups, globular or rounded and linear or elongated. The first group comprises oval, reniform or pyriform types. The latter includes cylindric, rectangular and vermiform types.

This method is also of value for the study of adjoining and related bony structures, such as the ethmoid and maxillary sinuses. Besides, visualization of the duct is helpful in choosing the method of operation and in the postoperative observation. The author shows that the method of Dupuy-Dutemps is preferable to the original Toti operation, and that the bony aperture, made in internal rhinostomy according to West-Pollack, is insufficient; also, that stricturotomy is to be abandoned, since the wound tends to close spontaneously. This happens especially when the duct is no longer probed and cicatricial tissue forms.

Of the many substances used, del Duca prefers iodized oil for its nonirritating qualities, because it requires no previous special preparation, and for its slightly antiseptic action on a sac containing mucus or a mucopurulent secretion.

For radiographic examination of the lacrimal passageways, the author prefers the occipitobuccal position, with the central ray directed 4 cm. above the occipital protuberance and passing through the root of the nose. This position displaces the shadows of the cranial base and the temporal bone downward, and gives a perfect view of the filled sac, thus permitting a study of the relations of the ethmoid and maxillary sinuses to the nasal bones and the walls of the orbit.

V. R. SYRACUSE.

Lens

HEREDITARY AND FAMILIAL CATARACT (NUMMULARY CATARACT, POSTERIOR CORTICO-NUCLEAR). P. VEIL AND A. FAVORY, Arch. d'opht. **47:** 666 (Oct.) 1930.

In this report the authors first point out some of the general characteristics of hereditary cataract. They draw attention especially to its occurrence in association with some other general disturbance referring to zonular cataract, with rickets, the cataract of myotonic dystrophy, floriform cataract and the cataract that has been noted in scleroderma. They present the pedigree of a family known to have had cataract for five generations, nine members being afflicted. They examined six patients and describe the biomicroscopic picture. In none of their patients did a general examination reveal any other abnormality. The heredity is direct, continuous and similar, and the transmission seems to take place through the female. These cases seem to be in the class of mendelian dominants. The biomicroscopic appearance was essentially the same in all cases. It appeared as a posterior corticonuclear discoid cataract made up of stratified disks surrounded by a powdery halo.

S. B. MARLOW.

THE RELATION OF CATARACT TO THE ENDOCRINE GLANDS. FISCHER, Clinique, Paris **24:** 119, 1930.

Since Erdheim's fundamental investigations, it has been known that destruction of the parathyroid glands is able to cause a cataract. The occurrence of such cataracts of endocrine nature has since been con-

firmed repeatedly. Whereas in cases of tetany and spasmophilia the seat of the cataracts is subcapsular, it is for the most part supranuclear when there is dysfunction of the parathyroid glands, and also in cases of myotonic cataract. In cases of tetany and myotonia the cataracts are diseases of the endocrine system due to a pluriglandular deficiency associated with a sympathetic lesion. Presenile cataracts are also found in various dermatoses, dermoneuritides and sclerodermias, along with degeneration phenomena on the part of organs of ectodermal origin. They are met with in people affected by hypothyroidism, sexual troubles and disturbances of growth, hence troubles of the system of the endocrine glands associated with diseases of the vegetative system result. Although these forms of cataract deserve particular interest, another form, the so-called "puberty cataract," which is often overlooked and which for the most part does not at first interfere with the acuteness of vision, should not be omitted. This form appears only at the age of puberty, and it is due to a deficiency of the interstitial glands. The "puberty cataract" can be seen only through the dilated pupil; it is composed of small gray patches and is located in the cortex around the equator of the lens, which area is spared. The "puberty cataract" is identical with Vogt's coronary cataract and with the blue cataract of former authors. It is almost always bilateral and familial. That this form of cataract never appears prior to the age of puberty, becomes denser during the sexual maturity of the individual and develops into complete opacity of the lens during old age points distinctly to a hormonal deficiency of the puberty glands. People with "puberty cataract" showed sexual impotence, and even a complete absence of sexual desire. Phenomena of hypoparathyroidism, however, were not detected in them. The disturbance in the secretion of the interstitial cells and the consecutive acidosis provoke an alteration of the lens by means of hormonal imbalance. Along with the definite and complete evolution of the glands at puberty, the destructive process in the cells of the lens comes to an end. If, however, the hormonal balance is not restored in the system during growth, the opacification of the lens will progress. Later on in old age, when the activity of the interstitial cells is naturally depressed, the cataractous destruction of the lens will reach its maximum. Horlacher said that in 60.5 per cent of the cases coronary cataract is a form of senile cataract. By putting this conception into practice, several authors have obtained reactivation of the puberty glands by Steinach's vasectomy or by Voronoff's procedure in initial cataract, and have thus achieved a clearing up of the tissue of the lens. "Puberty cataract" can be considered a pathognomonic sign of hormonal imbalance. At the age of puberty, cataract should be repeatedly searched for, and if it is found, vasoligation should be advised in order to activate the internal secretion of the gonads and thereby exert a beneficial therapeutic influence on the cataract. Vasoligation is not a severe operation; it is sufficient to perform it on only one side.

N. BLATT.

Lids

CONDYLOMATA AT THE INNER CANTHI. E. B. GRESSER, Am. J. Ophth. **13:** 886 (Oct.) 1930.

A woman, aged 39, presented a fading roseola on the body, annular syphilids about the mouth, condylomas of the inner canthi and naso-

labial junction and "split pea" papules at the angles of the mouth. There were also condylomas in the genito-anal regions, at the umbilicus, under both breasts, in the axillae and in the folds of the elbows. Mucous patches were present in the throat. These conditions were of one month's duration. The Wassermann reaction was 4 plus. The eyeballs were not affected externally or internally. W. S. REESE.

Methods of Examination

DETERMINATION OF THE RIGIDITY OF THE CENTRAL RETINAL ARTERY AND ITS CLINICAL IMPORTANCE. FRITZ, Arch. d'opht. **47:** 476 (July) 1930.

This paper is a preliminary report. The technic and rationale of plethysmo-oscillography is first described. Its principles are then applied to the retinal vessels through the use of Bailliart's dynometer. The results obtained in a variety of conditions are cited. The author feels that this is a particularly valuable procedure not only because of local disturbances of the vessels, but also because of the relationship which exists throughout the whole vascular system. The smaller blood vessels in the eye can be examined only by this procedure. It presents interesting possibilities. S. B. MARLOW.

Neurology

ABSENCE OF CHOKED DISK IN CEREBRAL TUMORS, AND RELATION BETWEEN CHOKED DISK AND ROENTGEN MANIFESTATIONS OF INTRACRANIAL HYPERTENSION. C. PEGORARO, Riv. di clin. med. **31:** 1177 (Nov. 30) 1930.

In a series of eighty-two cases of cerebral tumor, Pegoraro studied the frequency of choked disk and the roentgen manifestations of intracranial hypertension. From the ophthalmoscopic observations, he found that choked disk was absent in 20 per cent of the cases. The absence of this symptom was not found to be absolutely dependent on the volume or the nature of the tumor. He observed, however, a certain relation to the site of the tumor. He found choked disk frequent in tumors of the occipital, parietal and temporal lobes, of the semi-oval center, of the anterior cerebral fossa, of the cerebellum and of the hypophysis, but rare in tumors of Rolando's zone and of the hypophyseal infundibular region. The roentgen observations were less frequent than the ophthalmoscopic. This depended also, to some extent, on the site of the tumor but not on its volume and nature. The ophthalmoscopic and the roentgen manifestations of intracranial hypertension in the observed cases were not always associated. [J.A.M.A.]

Orbit, Eyeball and Accessory Sinuses

GIANT MUCOCELE OF THE FRONTAL SINUS. S. TRIOSSI, Rev. oto-neuro-oft. **8:** 63 (Jan.-Feb.) 1931.

Mucoceles of the accessory sinuses encroaching on the orbit are of interest to the oculist and the rhinologist. Triossi reviews a case of giant mucocele of the frontal sinus in a man of 40 and its diagnostic lessons.

Of the classic triad of clinical signs, a slowly progressive tumor at the inner angle of the orbit and exophthalmos downward and temporally with diplopia and a lessened acuity of vision were present. The third sign, which some investigators (Rollet) think is pathognomonic, a naso-orbital hyperostosis at the junction of the frontal, nasal, ethmoid and maxillary bones, causing a characteristic change of the root of the nose and of the facial expression, was absent.

The unusual secondary features were a noteworthy proptosis (not as extreme as in the cases reported by Garreau and Bellingham), a nonpalpable dehiscence of the floor of the sinus behind the orbital arch (Lyder-Borthen's sign), negative results of a rhinological examination and a history of a negative exploratory needle puncture. Transillumination showed a relative opacity of the left frontal sinus.

Careful roentgen projections, typical and special, showed a disappearance of the line corresponding to the left orbital margin, and, in the anterior portion of the orbital vault, a notable solution of the corresponding plate of the frontal bone continuing medially without clear boundaries. The left frontal sinus was clouded, and its supra-orbital recess was considerably dilated.

After a consideration of the differential diagnosis and by elimination, the author again considers the probability of a mucocele.

An ample superciliary orbitotomy was performed. On exposure of the periosteum, a second diagnostic puncture was performed, which yielded 25 cc. of a chocolate-colored mucoserous fluid containing large numbers of typical cholesterol crystals. Only then was the true diagnosis, which had hitherto remained uncertain, assured; hence the value of diagnostic puncture in questionable or difficult cases. The pathogenesis, however, could not be deduced from the history or from the clinical or operative observations. The nasofrontal duct was found impervious, but not due to neoplastic masses or to a process of condensing osteitis. As histologic examination of the mucosa of the frontal sinus was not possible, the theory of cystic degeneration of the mucosa remains without proof.

V. R. SYRACUSE.

CAVERNOUS ANGIOMA OF SIXTY-FOUR YEARS' STANDING; ROENTGEN DEMONSTRATION OF PHLEBOLITES. S. DE VRIES, Klin. Monatsbl. f. Augenh. **85:** 538 (Oct.) 1930.

A man, aged 64, gave a history of reduced vision and increasing exophthalmos since boyhood. He had refused operation about twenty years previously. Proptosis of about 3 cm. was present in one eye. The conjunctiva was greatly swollen. Keratitis and iritis made ophthalmoscopic examination impossible. The sensitiveness of the cornea was preserved; the globe was immovable. Vision was reduced to perception of the motion of the hand. A hard tumor could be felt between the globe and the upper orbital margin; the surface of the tumor showed a number of smooth prominences; pulsation was absent. The patient suffered from severe pain. A roentgenogram revealed the orbital walls to be intact; the orbit was enlarged, and in it were a number of isolated round shadows, which, later on, proved to be those of phlebolites. The globe and the tumor, which were enclosed in a thin capsule, were removed with the patient under local anesthesia, leaving the walls of

the orbit entirely smooth. Hemorrhage did not occur, and recovery was unhampered. The tumor, which measured 3 by 4 by 4 cm., consisted of a white, apparently fibrous substance, interspersed with rust-colored connective tissue and cavities of divers sizes up to that of a cherry; some of the cavities were empty; others were filled with blood. Very hard, pearly-white phlebolites could be lifted out readily. Their histologic structure was that of concentric calcifications, void of cellular elements. The walls of the arteries of the tumor were greatly thickened; obliterating endarteritis and calcifications were evident.

K. L. STOLL.

PERIODICALLY RECURRING UNILATERAL EXOPHTHALMOS: CASE REPORT. F. WULKOW, Klin. Monatsbl. f. Augenh. **85:** 669 (Nov. 28) 1930.

In 1922, in a man, aged 43, exophthalmos developed suddenly in the left eye without any known cause. He had never been sick, and his eyes had never shown any disorder. Vision was normal. No pathologic changes were found in the fundus of either eye. The exophthalmos disappeared after two days' rest. It recurred three years later after a paroxysm of coughing during an attack of grip, causing severe pain which at first suggested glaucoma. For several days the margin of the left disk was not clearly defined, after which it became normal, while the exophthalmos disappeared. The treatment consisted in rest in bed and a pressure bandage. A third attack, which occurred in 1927, ceased after a few days. It was noticed, however, that atrophy of the optic nerve and amaurosis had developed. In 1930, the patient returned with a severe manifestation of exophthalmos. It had developed after he had bent over to pick up a handkerchief. The left eye was turned slightly downward; there was no perception of light; the atrophy of the optic nerve was complete, and the anterior chamber was, perhaps, narrower than that of the right eye. The iris, lens, vitreous body and retina were free from changes. The intra-ocular tension was normal. The exophthalmos disappeared promptly.

Gummatous periostitis and other inflammatory processes could be ruled out as causative factors. The nose and adnexa were normal. Symptoms of thrombosis of the orbital veins, dermoid cysts and plexiform neuroma were excluded diagnostically. Exophthalmic goiter was not present. In the author's opinion, varicose veins were the cause of the exophthalmos.

K. L. STOLL.

RAPID CURE OF ORBITAL GUMMA. N. BLATT, Orvosi szemle **3:** 211, 1930.

Chronic inflammatory tumors of the orbit are fairly rare. Statistically, gumma of the orbit occurs in from 1 to 2 per cent of syphilitic diseases of the eye, and in 0.02 per cent of the total number of ophthalmologic cases. Such pathologic conditions seem to grow scarcer and scarcer, which fact is partly due to the modern antisyphilitic therapy, and partly to attenuation of the virulence of the syphilitic causative agent. Differential diagnosis must take especial account of tumors of the orbit, this being of particular importance for therapy. Diagnostic errors will more rarely be caused by diseases of the paranasal sinuses or

tuberculous diseases of the orbit. The area of predilection of orbital gummas is the superior wall of the orbit at the level of the superior orbital fissure. The most frequent clinical symptoms are exophthalmos, spontaneous pains appearing particularly at night, corneal anesthesia, motor disturbances of the muscles of the eyeball, palpebral edema, chemosis of the conjunctiva, iritis, optic neuritis and neuroparalytic keratitis.

The author describes a case of orbital gumma that occurred in a man, aged 38, who had had trouble with the left eye for three weeks. According to the history, he had had syphilis two years previously. He complained of intolerable headache starting from the left orbit. The right eye was normal; vision was 5/5. The left eye presented the following symptoms: tenderness of the left orbital wall, ptosis, slight exophthalmos, displacement of the eyeball inward, immobility of the bulb, exaggerated corneal reflex, Argyll Robertson pupil and optic neuritis. The visual field was normal; vision was 5/30. A roentgenogram showed an orbital tumor located on the lateral orbital wall. The Wassermann reaction was strongly positive. Therapeutic injections of mercury were followed by stomatitis and enteritis; arsphenamine produced exacerbation of the optic neuritis and impairment of vision. Cure and restitutio ad integrum were brought about by bismuth treatment, neobismosalvan being used, totaling twenty ampules injected at three or four day intervals. In alterations of the optic nerve in which arsphenamine and mercury therapy offer risks, bismuth therapy (neobismosalvan-bismuth, iodine, quinine and lecithin) proves harmless and efficient.

N. BLATT.

Pharmacology

THE HUMORAL MECHANISM OF STIMULATION OF THE THIRD NERVE. E. ENGELHART, Arch. f. d. ges. Physiol. **227**: 220, 1931.

An examination of the effects of aqueous humor of the rabbit and cat on the turtle's heart (*Bufo vulgaris*) failed to demonstrate the presence of "vagusstoff," i. e., acetylcholine. In rabbits, the application of three drops of 1 per cent physostigmime produced a substance in the aqueous similar in all respects to acetylcholine. If one eye was illuminated and the other kept in the dark, large amounts of this substance were found in the illuminated eye and practically none in the dark-adapted eye. A considerable quantity was found in the ciliary body and the iris, and the amount was increased by electrical stimulation of the third nerve. Following section and degeneration of the third nerve, the substance vanished completely from the ciliary body and the iris.

F. H. ADLER.

Retina and Optic Nerve

SIMPLE DETACHMENT OF THE RETINA: WITH A REPORT ON SEVENTY-FIVE CASES TREATED AT MOORFIELDS EYE HOSPITAL BY GONIN'S METHOD. J. H. DOGGART and C. D. SHAPLAND, Brit. J. Ophth. **15**: 257 (May) 1931.

The authors' definition of simple detachment of the retina is a partial or complete separation of the layer of rods and cones from the retinal pigment cell layer, arising either spontaneously or as the apparent result of a minor trauma to the globe from direct or indirect violence

A brief review of Gonin's views, the diagnosis, the significance and morphology of retinal holes, prognosis, topography, operation, after-treatment and resuls and four illustrative cases are given.

The operation as performed at Moorfields is described as follows: On the eve of operation, two marks are tattooed with india ink at diametrically opposite points of the limbus in line with the estimated position of the hole.

After the eye has been cocainized, about 0.5 cc. of a mixture of equal volume of 4 per cent procaine hydrochloride and 1 : 5,000 solution of epinephrine is injected beneath the ocular conjunctiva at the site determined for cautery puncture. After an interval of five minutes, a knotted guiding thread is passed through the episcleral tissue at the limbal pigment mark remote from the side of the retinal hole. The ocular conjunctiva is then divided at a point about 0.5 cm. from the pigment mark on the side adjacent to the retinal hole, and is incised for a length of about 1.5 cm., so that the incision is parallel to the tangent to the arc of the limbus at the pigment mark. Tenon's capsule is opened and the sclera bared over the required situation. The guiding thread is passed through a hole punched in the center of the blade of a Desmarres' retractor, which is placed in the conjunctival wound and retracted, thus giving access to a wide area of sclera. The guiding thread is then arranged so that it passes from the first tattoo mark across the center of the cornea over the second pigment spot, so that its prolongation crosses the site of the retinal hole. The predetermined distance of the retinal hole from the limbus is marked on the exposed sclera with a pair of callipers, one point of which has previously been dipped in sterilized india ink. Two or more conjunctival sutures are inserted in the cut ends of the ocular conjunctiva, and the guiding thread is withdrawn. An electric cautery at white heat then sears a way through the sclera at the point determined, and, as soon as the subretinal fluid has escaped, it is again heated to white heat and plunged through the opening in the sclera so made, and immediately withdrawn, the average depth of penetration being 1 cm., and the duration, two seconds. The conjunctival sutures are immediately tied off; the speculum is removed, and double bandages are applied.

During 1930, seventy-five patients with retinal detachment were submitted to the operation of cautery puncture by Gonin's method at Moorfields Eye Hospital. Of these seventy-five, twenty-four were discharged with the retinal detachment back and the visual field full; twelve showed improvement either in the visual field or in visual acuity; the remaining thirty-nine were unchanged or made worse. To obtain 32 per cent of cures and 16 per cent of improvements in such a serious and hitherto almost hopeless condition as detachment of the retina is indeed striking, especially as the method was entirely new, and but little attempt was made to select cases during the first six months of the year.

W. ZENTMAYER.

OBLITERATING CAUTERY PUNCTURE IN DETACHED RETINA. J. GONIN, Ann. d'ocul. **168**: 1 (Jan.) 1931.

Gonin begins his article with a short history of the operation with which he first corrected a case of spontaneous retinal detachment by thermocauterization in 1916. He reported cases from year to year to

May, 1930, when he published a report of the cases of 240 patients with spontaneous retinal detachment, the majority of whom were treated by operation.

Gonin emphasizes the constancy of retinal tears and the rôle they play in detachment. He also says that one must obtain a cessation of the outflow of vitreous at the earliest possible moment, which, forming the retroretinal effusion, maintains the detachment.

In his examination of the eye, Gonin obtains maximum mydriasis from cocaine and homatropine, and he uses the indirect method with the ophthalmoscope. He directs his examination first to the section of the retina that corresponds to the defect in the visual field complained of by the patient. He emphasizes, however, that there may be other tears or holes in totally different areas.

The appearance of the fundus in regard to the appearance of the tears, their position, etc., is accurately drawn on a chart. Completion of the chart and the measurements necessary take considerable time, according to the assistance given by the patient. When the possibility of operation has been established, it is necessary to mark on the external surface of the eye the meridian and parallels corresponding to the tear, that is, the direction and the distance calculated from the border of the cornea to the point in the sclera where the cautery puncture is to be made. Gonin does this marking on the globe, which is indispensable, with a solution of alcohol and gentian violet retouched with China ink. After the direction of the tear has been indicated, it is necessary to establish the distance from the tear to the ora serrata.

The article is divided into the following sections: (1) history and preliminary examination of the diseased eye; (2) the finding of the retinal tear and its localization with the aid of an ophthalmic chart; (3) the repair of the tear on the ocular globe; (4) the preparation for operation; (5) the operation; (6) postoperative precautions; (7) repeated operations, and (8) the instruments.

S. Hanford McKee.

Retrobulbar Neuritis of Tuberculous Origin. C. Mariotti and G. Laschi, Arch. di ottal. **38**: 17 (Jan.) 1931.

The authors review the literature on the causes of retrobulbar neuritis. According to Bergmeister, the optic nerve may be affected with tuberculosis from a focus in the globe, orbit or meninges, and also without any involvement of these structures. A number of cases of the latter condition are cited, and the authors believe that we must accept the existence of a primary retrobulbar neuritis due to tuberculosis.

They report the case of a woman of 30 who complained of loss of vision. This had been diagnosed as the result of a pituitary tumor, and three courses of irradiation to the sella resulted in improvement of vision, followed each time by relapse and further progress of the condition. When the patient was seen in the eye clinic five years after the beginning of symptoms, vision was 2/10 in the right eye and 2/50 in the left, and there was concentric contraction of the fields.

X-ray pictures of the sella were normal, but an increased density in the lesser wing of the sphenoid was noted. The chest showed signs of previous pleurisy and some apical pathologic change.

The tuberculin test was markedly positive, an intradermal injection producing a local, focal and general reaction. Under intravenous injection of calcium and irradiation of the sellar region with a view to reaching the supposed tuberculous osteitis, vision improved to 2/10 in the right eye and 1/10 in the left. A course of treatment with tuberculin was being given. An attack of anterior uveitis brought on by the first injection cleared up completely.

The value of a careful interpretation of the x-ray picture is emphasized, the authors being able by this means to decide against pituitary tumor and in favor of an osteitis of the sphenoid. It is believed that roentgen therapy was of definite value in attacking the supposed tuberculous process, and that the possibilities of such therapy in retrobulbar neuritis have not been sufficiently considered by ophthalmologists. (Bibliography.)

S. R. GIFFORD.

THE INFLUENCE OF SYMPATHETIC STIMULATION ON THE RETINA. S. DUCRET AND S. KOGO, Arch. f. d. ges. Physiol. **227:** 71, 1931.

The authors performed experiments on the frog's retina to prove the rôle of the vegetative nervous system in the state of excitation of the organs (see Hess, W. R., and Lehmann, F. E.: *Arch. f. d. ges. Physiol.* **211:** 603, 1926). Following unilateral sympathicotomy, histologic studies were made on the shape and position of the rods and cones in the two retinas. In spite of different intervals between sympathicotomy and enucleation of the eyes, the authors did not succeed in demonstrating any differences between the two retinas either in the light or the dark adapted condition.

A third series was examined in which the eyes were given a moderate exposure to light just prior to enucleation. The results in this series were also negative.

F. H. ADLER.

CHOKED DISK CAUSED BY HYPOTENSION. A. EPPENSTEIN, Klin. Monatsbl. f. Augenh. **85:** 672 (Nov. 28) 1930.

A man, aged 79, suffered from an acute attack of inflammatory glaucoma in one eye; this eye had been always weaker than the other one. The tension was 28 mm. in the affected eye and 12 mm. in the other. Vibratory massage eliminated the severe pain, reduced the tension to 10 mm., and increased the vision slightly. The pupil could not be contracted with pilocarpine, although the tension dropped below the normal point, to 5 mm. The fundus was normal at first, but a week after the attack the disk showed some prominence. Edema of the retina appeared, and the central retinal vessels were strongly filled. About a month later the fundus was again normal. The pupil remained slightly irregular, but its reaction had returned. The author explains the mechanism producing the choked disk as a consequence of a pathologic lack of resistance of the tissues of the disk.

K. L. STOLL.

Trachoma

CHRONIC HYPERPLASIAS OF THE CONJUNCTIVA AND TRUE TRACHOMA. C. PASCHEFF, Arch. d'opht. **47:** 821 (Dec.) 1930.

Pascheff points out that in spite of much work, both old and new, there is as yet no entirely complete understanding of the exact nature of

trachoma. This paper is an attempt to unify the conceptions of its nature. The whole subject of chronic hyperplasia of the conjunctiva is reviewed under a histologic classification which groups them as: (1) plasma cell hyperplasias, (2) lymphocytic hyperplasias, (3) follicular hyperplasias, (4) papillary hyperplasias and (5) hyalinoid hyperplasias.

The first group concerns plasmomas. The second is subdivided into four groups: (*a*) aleukemic lymphomatous hyperplasias, (*b*) leukemic lymphocytic hyperplasias, (*c*) metastatic lymphocytic hyperplasias, (*d*) local lymphocytic hyperplasias. The third group is subdivided into: (*a*) simple follicular hyperplasia, (*b*) miliary follicular hyperplasia, (*c*) confluent follicular hyperplasia. This group is described in detail. The fourth group is subdivided into three types: (*a*) simple catarrhal, (*b*) lymphopapillary, (*c*) fibropapillary. In this last group he places spring catarrh. The fifth group is divided into primary and secondary types. Trachoma, according to Pascheff, falls into the large groups of follicular hyperplasias, especially in the confluent follicular and folliculomatous types. He believes that those who are searching for a causative organism err in denying the follicular character of true trachoma. True trachoma, he believes, is a highly differential lympho-adenoid tissue. The most favorable terrain for the development of trachoma is lymphatism.

S. B. MARLOW.

Tumors

DIFFUSE NEUROFIBROMATOSIS WITH PROPTOSIS. R. FOSTER MOORE, Brit. J. Ophth. **15:** 272 (May) 1931.

One case is reported in detail, and particular points in three other cases seen by the author are referred to.

The patient was a man, aged 33. At the age of about 6 months, the right eye became prominent following a fall downstairs. At 6 years, partial resection of the orbital wall and ligation of the carotid artery had been performed. Scattered over the body were subcutaneous and intradermal nerve tumors. The skull was brachycephalic. The right orbit was considerably larger than the left, and no details of the posterior orbital wall were seen in a roentgenogram. The right eye was displaced downward and forward. It was possible to insert one's finger far enough behind the globe to feel the optic nerve. The lids closed well over the eye. Pulsation could be felt in the closed lids; it was not of the expansile type. There was no bruit, and the proptosis was not reducible. Movements of the globe were but little restricted. The fundus was normal. The visual acuity was 6/9, and the visual field was full.

The points of interest in this condition are: the pulsatile nature of the proptosis, which occurred in all four of the author's cases; it seems evident that the pulsation that is felt is not of direct vascular origin but is transmitted directly to the orbital tissues from the brain; fulness and bulging in the temporal fossa; in one case a trephine operation had been performed over this area, perhaps with the expectation of finding a tumor; the presence of opaque nerve fibers in the retina of the left eye may have been a fortuitous occurrence, but on the other hand it seems likely that they are in some way connected with the abnormality of the other nerves of the body.

W. ZENTMAYER.

Sympathetic Ophthalmia

SYMPATHETIC OPHTHALMITIS DEVELOPING FORTY YEARS AFTER AN OPERATION ON THE FELLOW EYE. P. SANDER, Brit. J. Ophth. **15:** 25 (Jan.) 1931.

A woman, aged 48, gave a history of having lost the sight of the right eye after an operation. The left eye was a source of trouble in her youth. From time to time it became painful and red, and the sight failed. Examination showed on the right side an atrophic shrunken globe with corneal leukoma. There was ciliary tenderness. The eye was blind. The left eye was trachomatous and exhibited signs of iridocyclitis with tenderness. The visual acuity equaled 6/18. The right eye was enucleated and found to contain bone. Under treatment, the media cleared disclosing papillitis. After three months the eye was quiet. Visual acuity equaled 6/12.

W. ZENTMAYER.

Therapeutics

AIR INSUFFLATION INTO THE ANTERIOR CHAMBER. L. SALLMANN, Nat. M. J., China **17:** 6 (Feb.) 1931.

Certain protective substances circulating in the blood are prevented from entering the ocular fluids by the so-called hemato-ophthalmic barrier situated in the walls of the intra-ocular capillaries. To break down this barrier is frequently of importance, and many methods, notably paracentesis of the anterior chamber and subconjunctival injections of saline, have been employed to obtain this result. Sallmann set out to find a method to facilitate the passage of remedies from the blood stream into the eye, and subsequently to compare the efficacy of this procedure to that of the other methods used for the same purpose. He injected fluorescein intravenously, withdrew the aqueous, and replaced the latter with air. He then studied the appearance of the fluorescein in the anterior chamber. By a modification of an ingenious device of Lindner's, he was able to measure the concentration in amounts as small as 1:8,000,000. He experimented first without and then with the air injections and compared the relative values of insufflation, paracentesis and subconjunctival injections of 10 per cent saline. He concluded that air insufflation enabled him to break through the hemato-ophthalmic barrier to a remarkable degree and for a long time, and that this method was superior to the other procedures.

G. M. BRUCE.

RADIUM TREATMENT FOR EPITHELIOMAS OF THE EYELID. M. APPELMANS, Rev. belge sc. méd. **3:** 829 (Dec.) 1930.

Appelmans describes a method of treatment with radium for epitheliomas of the eyelids which he used in seventy-five cases. Although the cases are too recent to ascertain the end-results, the immediate results of the treatment were excellent from both the functional and the esthetic standpoints. The method employed is a follows: 1. In cases of small epitheliomas that are merely intradermal, that leave intact the function of the eyelid and that have not invaded the soft tissues of the orbit, the treatment consists of direct application of tubes con-

taining 2 mg. of the element radium, 12 mm. long filtered with 1 mm. of platinum. The tubes are placed not more than 1 cm. apart, axis to axis, so that they cover the entire tumor and a little of the surrounding tissues. They are held in place by narrow strips of sparadrap. The dressing is renewed every day, and the applications are continued for seventy-two hours. 2. Since small epitheliomas of the inner corner of the eye are often deeper than they seem, invading the soft tissues of the orbit, if one is not entirely certain as to the superficiality of the tumor it is advisable to use a molded applicator placed 3 cm. away or to employ telecurietherapy with a fixed applicator, placed from 5 to 6 cm. away. When a molded applicator is used, it is necessary to destroy 1.5 millicuries of radium emanation per square centimeter of the radiant surface, which requires a dose of 100 mg. of radium per square centimeter, filtered with 1 mm. of platinum and spread as regularly as possible on the surface of the applicator. 3. In case of epitheliomas that have invaded the soft tissues of the orbit, when there is still a chance of saving the eye, one may use telecurietherapy, a molded applicator or a combined method consisting of radium puncture, followed by a molded applicator or by telecurietherapy. For the method of radium puncture one uses needles of mild strength (1 mg. for each 1.5 cm. of radiant length, for example) filtered with 0.5 mm. of platinum. The needles, placed 1 cm. apart, may be left in place five days. Afterward 0.5 millicurie or radium emanation per square centimeter of radium surface should be given by means of a molded applicator. When the eye is too deeply involved to be saved, it is advisable to make an evisceration of the orbit with a diathermic loop or knife, followed by coagulation of the entire cavity. A dressing of 2 mg. tubes of radium may then be applied for thirty-six hours. [J.A.M.A.]

BISMUTH THERAPY IN OPHTHALMOLOGY. B. I. CHETVERIKOVA, Russk. Opht. J. **12**: 577 (Nov.-Dec) 1930.

The author treated twenty-seven syphilitic patients with biochinol, the Russian preparation of bismuth. Biochinol consists of 20 per cent of bismuth, 48 per cent of iodine, 31 per cent of quinine and 10 per cent of oil. There were sixteen cases of keratitis parenchymatosa, eight cases of optic atrophy, one of embolism of the central retinal artery, one of optic neuritis and one of paresis of the superior rectus muscle. The injections were given intramuscularly, every two to three days, the initial dose for children being 0.5 cc., and for adults 1 cc. This dose would be increased to 1 and 2 cc., respectively, on consecutive injections. The author believes that this dose is sufficient since the smallest doses of bismuth are highly spirocheticidal. Each course of treatment consisted of from fifteen to twenty injections. No stomatitis was observed during the treatment. The urine was examined once in from seven to ten days. Albumin and erythrocytes were found in four patients; two patients showed anemia and an increase of the leukocytes. The Wassermann reaction before the treatment was four plus in twenty-two patients, three plus in two patients and two plus in three patients. Five patients, i. e., 30 per cent, gave a negative result after the first course of treatment, and the blood remained unchanged in nine patients, or 50 per cent. Three of the last group received combined treatment of neoarsphena-

mine and biochinol, but the Wassermann reaction remained positive. The vision improved from 0.05 to 0.2 in seventeen patients with keratitis parenchymatosa, from 0.1 to 1 in one patient with optic neuritis, and in all the other patients there was only slight improvement.

The author draws the following conclusions:

1. Bismuth, though not a perfect remedy, can be recommended highly in the treatment for syphilis with ocular involvements.
2. No local or general complications were observed when small doses were used.
3. Bismuth is not provocative, as in most of the cases with the keratitis parenchymatosa it prevented the involvement of the second eye.
4. When compared with injections of neoarsphenamine, bismuth has the advantage of a more simple technic.
5. Patients who, out of embarrassment, would not go to a venereologist, are benefited by the fact that any oculist can undertake the injections at his office.
6. Optic atrophy should be treated by bismuth, as the process is retarded.
7. If the Wassermann reaction is positive after one course of injections, a combined treament of bismuth with neoarsphenamine should follow.

O. SITCHEVSKA.

Toxic Amblyopia

TWO CASES OF QUININE AMBLYOPIA WITH UNUSUAL OPHTHALMOSCOPIC PICTURE. J. N. DUGGAN and B. P. NANAVATI, Brit. J. Ophth. **15:** 160 (March) 1931.

The first case was that of a girl, aged 9 years. For an acute attack of malaria she had taken four doses of 1½ grains of quinine at four hour intervals the first day and two doses the following day, when she suddenly went blind in both eyes. There were no other symptoms of quinine poisoning. Twenty hours after the onset, the eyes were still totally blind. In both eyes the optic papilla and retinal vessels were normal. There was edema of the retina extending to the periphery of the fundus which looked yellowish white. A faint red spot marked the macula. Four days later the papillae were white, the retinal vessels were much contracted, the fundus reflex was normal, and both macular regions showed glistening yellow-white dots arranged irregularly around the macula. One week later there was perception of light. The retinal vessels were more contracted but the macular changes were less marked. Seventeen days later, vision was 6/6. Each field was concentrically contracted to within 10 degrees of fixation. The papillae remained white, and the vessels were contracted with white lines along them.

The second case was that of a man 20 years of age. He had taken 378 grains in 2 grain doses spread over a period of three weeks. The onset of blindness was sudden. The fundus picture, except for the absence of the macular glistening spots, was the same as in the first case. In seventeen days vision had returned to normal.

Since malaria is so common in the tropics and quinine is so freely used, the author is of the opinion that some of the cases of optic atrophy in which no definite cause can be found might be due to the toxicity of quinine.

W. ZENTMAYER.

A Case of Quinine Amblyopia, with a Central Colour Scotoma of One Eye and Total Blindness of the Other. J. N. Duggan and B. P. Nanavati, Brit. J. Ophth. **15:** 164 (March) 1931.

A woman, aged 25, had taken about 5 ounces of quinine at one dose; marked toxemia resulted, accompanied by unconsciousness. On recovering consciousness, she was totally blind in both eyes. Vision was recovered equally in the two eyes, but later the right became again suddenly blind. On examination, the patient was able to see movements of the hand with the right eye, and in the left, with the myopia corrected, vision equaled 6/12.

In both eyes there was optic atrophy and contracted retinal vessels with white lines along them. The field of vision for the left eye was irregularly contracted within 20 degrees of fixation, except on the nasal side its limits almost reached fixation. With "tiny" test objects, a definite scotoma for green and an indefinite one for red were present at fixation.

The authors believe the presence of these scotomas supports the theory that quinine directly attacks the ganglion cells of the retina with resulting ascending degeneration, although it is quite probable that the degeneration of the nerve fibers is primarily due to lack of circulation.

W. Zentmayer.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

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April 20, 1931

A CASE OF VOLUNTARY NYSTAGMUS. DR. HUGH S. MCKEOWN (by invitation).

R. H., a boy, aged 13, was admitted to the Vanderbilt Eye Clinic in December, 1930. He had discovered, about one year previously, that he was able to "make his eyes dance." Examination showed a mild myopia, and the vision was normal with correcting lenses. There were no other positive findings. The boy could initiate or arrest at will rapid horizontal nystagmoid movements of both eyes, which were regular, rhythmic and synchronous. Analysis of these movements showed them to be of equal components, of about 6 mm. excursions, divided equally to the right and to the left about the primary position. Vertical nystagmus could not be produced. The boy had learned to use the antagonistic muscles in the associated movements of eyes to the right and to the left, producing a tremor similar to that produced in all parts of the body when muscles are rapidly alternately contracted as antagonists. The mechanism of its production probably lies in the conscious control of the supranuclear centers governing the associated movements of the eyes in the horizontal plane.

DISCUSSION

DR. A. EDWARD DAVIS: (Dr. Davis reported a case of a similar nature in which the patient had been under observation for thirty-four years; typical visual horizontal nystagmus, with very short motions to the right and left were produced at will.)

DR. MARTIN COHEN: Why can the patient produce horizontal and not vertical nystagmus?

DR. H. S. MCKEOWN: Probably he has never tried to produce vertical motions.

AN OPERATION FOR EPIPHORA. DR. BENJAMIN FRIEDMAN (by invitation).

An elderly man presented himself at the Knapp Memorial Eye Hospital on the service of Dr. Grout with the history of tearing which was exceedingly disturbing at times. There was a slight ectropion of the lower lid of each eye. In addition, the puncta were placed farther than usual from the inner margins of the lids. Both lacrimal ducts allowed

the free passage of fluid through the nose. A lacrimal probe was inserted in the lower canaliculus as far as the bony portion. A 2 mm. trephine was placed against the mucous surface of the lid, and by rotating it against the probe, a button of tissue was excised from the inner surface of the lid about 3 mm. distal to the natural ostium. The new opening was kept patent for several days by means of a thread passed into the natural punctum and out through the artificial one. After several months the patient was almost entirely relieved of his tearing. The operation seems to give best results in cases of atresia of the punctum in which repeated dilatations are of no avail.

SUBCONJUNCTIVAL HEMORRHAGE AS A DIAGNOSTIC SIGN IN INTRA-OCULAR FOREIGN BODIES. DR. BENJAMIN FRIEDMAN (by invitation).

This article will appear in full in a later issue of the ARCHIVES.

DISCUSSION

DR. MARTIN COHEN: I should like to ask if it would be possible to locate the foreign body if a roentgenogram had been taken. Might it not have been localized in the subconjunctival area?

DR. BENJAMIN FRIEDMAN: Evidently my object in presenting this case was not made clear. I wished to call attention to the fact that a roentgenogram was not necessary; the presence of the hemorrhage in the nasal area was sufficient proof that the foreign body had reperforated the eyeball and lodged under the conjunctiva.

A SIMPLIFIED AND ABBREVIATED METHOD OF MUSCLE RESECTION. DR. SIGMUND A. AGATSTON.

By the method described by Dr. Agatston, it was claimed that traction, the cause of most discomfort, is avoided. It is a simple resection, with suture through the stump of the muscle. The sclera under the stump and the conjunctiva on the corneal side are included.

DISCUSSION

DR. CONRAD BERENS: Posey and Lancaster have used this method for years in resecting a muscle, and the operation that I described in 1925 (*Tr. Ophth. Soc. U. Kingdom* **45:** 415, 1925) differs principally in the incision in the conjunctiva, which is made as close to the semilunar fold as possible; the wound is then closed with a running suture. With this method I have had no infections, and granulations are rare. The two double-arm sutures are a great improvement over the old Reese method of resection, because with the older operation the conjunctiva was turned in at the old insertion, and granulating wounds and infection sometimes resulted.

THE DEVELOPMENT OF THE CORNEA IN THE CHICK EMBRYO. DR. GEORGE F. LAIDLAW.

The growth of the cornea from its simplest form, beginning with the 3 day old chick to its full development, was traced by means of lantern slides. In the beginning the cornea consists of a double layer

of ectodermic epithelium. At this time the lens is already formed. The next stage, that of the 4 day old chick, clearly shows the different parts played by the ectoderm and mesoderm in the construction of the cornea. The foundation of the future cornea seems to be in a homogeneous substance, which is apparently excreted by the ectodermic epithelium. The posterior endothelium springs from the mesenchymal cells. They later invade the cornea and become the corneal corpuscles, laying down the connective tissue fibers of the finished cornea. Bowman's and Descemet's membranes seem to be vestiges of substance exuded from the ectodermic epithelium. In the chick these membranes are collagenous and not elastic tissue. The cornea is formed in a manner similar to the skin of a tadpole. At first there is a collagenous exudation from the ectodermic epithelium, and this is later invaded by cells from the mesenchyme. In the eyes of a 12 day old chick, stained with my silver stain, it was found that cells of ectodermic and mesodermic origin could be differentiated; those from the ectoderm stain positively, while those from the mesoderm stain negatively or are invisible. They can be made visible again by counterstaining. Tumors springing from these cells retain their positivity or negativity to the silver stain.

DISCUSSION

DR. ALGERNON B. REESE: It seems to me that Descemet's membrane and Bowman's membrane cannot be classified as similar structures. The former, that is, Descemet's membrane, takes the elastic tissue stain; it retracts and curls when cut; it is a cuticula product and regenerates, whereas Bowman's membrane has characteristics other than those enumerated.

I did not quite understand the fate of this homogeneous collagen which appeared at about the third or fourth day. What counterpart does it have in the adult cornea, or does it disappear entirely?

DR. DANIEL B. KIRBY: I think that Dr. Laidlaw may possibly have the answer to the question whether the so-called melanosarcoma is ectodermal or mesodermal in origin. There is quite a discussion as to whether or not mesodermal cells can really produce pigment, and I think that if this silver method of Dr. Laidlaw's is followed, the answer may be found.

DR. BENJAMIN FRIEDMAN: There is one question that interests me. In certain chosen sections it is possible to demonstrate fibrillar adhesions between the surface ectoderm and the lens. Similar adhesions were described first by Lenhossek between the lens plate and the retina. The fibrillar structures between the surface ectoderm and the developing lens have been designated as the anterior vitreous. In man they persist only for a short time, but in birds (in the chick, for instance) they persist for a longer period; they are present in greater quantities, and certain writers have ascribed to these cells of the anterior vitreous a function that is related to the formation of Descemet's membrane and Bowman's membrane. They claim that this anterior vitreous possibly lays down the hyaline membranes of the cornea. Has Dr. Laidlaw been able to demonstrate these adhesions? If so, does he find the relationship mentioned?

Another point to recall is that the eye of the chick in particular is very poor in mesodermal cells. If one looks at the vitreous, one will find such a paucity of mesoderm that it is difficult to detect the cells; similarly, a scarcity of mesoderm is evident in the region between the lens and the ectoderm. In the human being, however, a different condition prevails. Seefelder says that in none of his sections was he able to find the total absence of mesoderm between the lens and the ectoderm. The mesoderm in man is present to a far greater extent, and one should not draw an unqualified parallel between the cornea of the chick and the human cornea, regarding the proportion and time of appearance of mesoderm and collagen in each.

DR. GEORGE F. LAIDLAW: We cannot transfer conclusions from one species to another. The formation of the mammalian cornea is not necessarily that of the chick. However, in Kessler's book, published in 1877, are pictured the formation of the cornea in the chick, the amphibian and the sheep. The eyes were fixed in osmic acid and stained with carmine, the usual technic in those days. Kessler described and illustrated the formation of the cornea of the sheep exactly as I have shown you in the chick. At first there is nothing but a layer of ectodermic epithelium; then a homogeneous substance oozes out from the epithelium. Later this homogeneous substance is peopled with fibroblasts.

As far as the anterior vitreous is concerned, there is not a sign of it in these sections. There is nothing between the cornea and the lens. There is plenty of mesenchyme on each side of the cornea, but it does not grow between the cornea and the lens until the homogeneous blue collagen has oozed out of the epithelia.

Dr. Reese asked about Descemet's membrane. There is considerable elastic tissue in the deeper layers of the cornea, but in the chick Descemet's membrane stains like collagen just beneath the endothelium, and the elastic tissue is in the substance of the cornea beneath that. I do not know what becomes of the collagen. On the fourth or fifth day, the blue layer appears. As it becomes peopled with fibroblasts, the color fades. That may be because it is being drawn out into finer threads. The thickness has a great influence on the color. I do not know if it disappears and the fibroblasts lay down collagen from some other surrounding fluid. Just where collagen comes from is a much discussed question. If we knew how a homogeneous substance is converted to a collagen fiber, we should know all about the formation of fibrous tissue, and we do not know that.

DR. CONRAD BERENS: I should like to ask Dr. Laidlaw if he has published his silver technic.

DR. G. F. LAIDLAW: Yes, in the *American Journal of Pathology* (5: 239 [May] 1929).

DR. BERENS: Have you studied the so-called glioma of the retina with your silver stain? There is controversy as to its mesoblastic or epiblastic origin.

DR. G. F. LAIDLAW: We are studying malignant tumors of the eye, but we have not reached definite conclusions. The origin of these tumors involves the embryology of the nervous system, and this is still obscure at many points. For instance, both the choroid of the eye and the pia of the brain are supposed to come from the mesoderm. Yet the cells

of melanoma of the eye and of meningioma of the pia stain positively with the silver stain, like ectodermic cells. Why should the pia and its representative in the eye, the choroid, persist in producing tumors that stain positively? This brings us to the French pathologists, Masson and Oberling. In the chick embryo they demonstrated, to their own satisfaction at least, that the pia is of ectodermic origin. Then with this selective silver stain, I show that pial and choroidal tumors stain like ectodermic cells. This is the situation so far.

SIGNIFICANT HEMORRHAGIC RETINAL LESIONS OCCURRING IN BACTERIAL ENDOCARDITIS—ROTH'S SPOTS. DR. WILLIAM BROWN DOHERTY and DR. MAX TRUBEK (by invitation).

Dr. Doherty and Dr. Trubek described what they believe is a typical retinal lesion occurring in bacterial endocarditis. The following conclusions are reached:

1. The characteristic elliptical retinal hemorrhages with white centers occur in the bacterial endocarditides, acute and subacute, and in the severe anemias, notably pernicious anemia.

2. The discovery of this lesion, because of its significant appearance, may aid in early diagnosis.

3. The lesion occurs in both eyes, with a little greater frequency in the left in our series.

4. The lesion has little prognostic value in subacute bacterial endocarditis; in several instances it had appeared and disappeared in successive crops many months before death.

5. We suggest that after further study, the designation of retinitis of endocarditis might be appropriately applied.

DISCUSSION

DR. ALEXANDER LAMBERT: So far we have only found these lesions in malignant endocarditis, and they are of great value when found.

DR. MARTIN COHEN: I do not believe that these lesions are characteristic of endocarditis. They are seen also in leukemia, polycythemia and other conditions. Hemorrhagic spots in the conjunctiva are much more pathognomonic.

DR. SIGMUND A. AGATSTON: From the description of the lesions, it would seem as though they must be thrombotic lesions of the minute veins. They are hardly like emboli. The lesion is so small as to cause a small spindle-shaped hemorrhage, on account of the nerve fibers crowding around, which would make it appear as though the lesion were a thrombus in a small venule with resulting hemorrhage. An embolism usually does not result in hemorrhage unless it is septic and causes real abscess formation. The small lesions would rather correspond to a thrombotic process resulting in localized hemorrhage and possibly a localized inflammatory reaction with leukocyte collection.

DR. BERNARD SAMUELS: I have seen quite a number of these hemorrhages with the white centers, at the New York Hospital. More than once the interns have informed me that the patients were suffering from malignant endocarditis. I think, however, much the same picture is seen, as Dr. Martin Cohen said, in both anemias and malignant endocarditis.

The changes in the fundus themselves are not pathognomonic for malignant endocarditis, that is certain, but they are very suggestive. They must be correlated with the blood picture, and if this is positive, then the diagnosis of malignant endocarditis is complete.

DR. W. B. DOHERTY: I think that if I were to make a drawing of each one of the three lesions, as Dr. Trubek and I have seen them, I could impress you by what we are trying to tell you. In endocarditis the lesion occurs in the superficial layer. The lesion assumes a position corresponding to the nerve fibers, and it appears and disappears rapidly, without leaving any trace of its former presence. It has a distinct outline with a characteristic center. That is the picture in bacterial endocarditis. In anemias, I will admit that there is a considerable similarity, but on careful observation I think you will find that the hemorrhage will appear slightly darker, and if you focus carefully on it, you can see the refractile striae of the nerve fibril layer. I think that is the differentiation—that you can see the refractile striae, and the hemorrhage seems to accentuate the presence of the nerve fibers. You cannot mistake them for the lesions of leukemia because in that condition the hemorrhages assume an irregular outline with faded borders and a yellow center. Not only that, but you have a distinct yellowish background with a marked tortuosity of the vessels. It is impossible to confuse endocarditis with either anemia or leukemia.

May 18, 1931

A MODIFIED MOTAIS OPERATION. DR. DANIEL B. KIRBY.

A girl, aged 19, with congenital ptosis, had been unsuccessfully operated on elsewhere at the age of 3. The palpebral fissures measured 4 and 3 mm. They were narrower in elevation than in depression, showing that the lid did not follow the globe when looking upward. Dr. Wheeler has emphasized the fact that this finding is a direct indication for the Motais or superior rectus transplantation type of operation. The original Motais operation is performed through the conjunctiva. This has been modified by Shoemaker, who uses a second incision combined with the conjunctiva, and I do the entire operation through a second incision. The incision is made 2 mm. below the level of the upper border of the tarsus. The dissection may be facilitated by shifting the speculum (Meller) into the different layers as the operation progresses—the skin, the levator, the two layers of Tenon's capsule. In fashioning the tongue of muscle, a fine straight surgical probe beneath the muscle is useful instead of the hook; then a single pass of the suture, a thin knife to start the tongue at the sclera, and the de Wecker scissors to cut it backward. Any excess tissue in the lower field is removed, as there is no object in putting any extra load on the slender muscle tongue. The muscle tongue suture is brought out through the margin of the lid. A secondary suture of silk is then used to place the muscle in firm apposition with the epitarsal tissue and

is tied after being passed through the skin. One feature that proved troublesome for a long time and in one case produced a temporary entropion was the pushing down of the skin in irregular folds over the area of the epitarsal dissection. This has been prevented by putting in double-armed sutures which cause the skin to be smoothed out below and to adhere to the epitarsal tissue in the proper place. In regard to the protection of the cornea immediately after the operation, the insertion of two intermarginal lid sutures, about 5 mm. from each of the canthi, has narrowed the palpebral fissure satisfactorily, protecting the cornea by lifting the lower lid and not putting any undue strain on the upper lid.

TWO CASES OF EXENTERATION OF THE ORBIT WITH SKIN GRAFTS. DR. DANIEL B. KIRBY.

CASE 1.—A woman, aged 53, had a squamous cell epithelioma of thirty-one years' standing for which she had received considerable treatment with radium, the x-rays, caustics and plastic surgery, but which in the end necessitated exenteration.

CASE 2.—A man, aged 73, had a melanosarcoma that originated in the globe; it perforated and produced a large mass in the orbit, metastasizing to the liver. He gave a history of a fall three years before examination. Examination showed detachment of the retina. Two years later the globe started to protrude. A roentgenogram showed a shadow in the left antrum, and a diagnosis of sarcoma was made. Evidence of metastasis of the liver was present. To avoid the development of an ulcerating fungating mass, exenteration was decided on. The antrum was opened, and a cystic condition was found with ordinary polypi.

In regard to the technic, the suction tip I described for use in operations on the lacrimal sac proved useful in removing the blood from the orbit of the cavity. In taking the graft from the leg, it is better to use oil, such as liquid petrolatum or white petrolatum, on the knife in order to allow it to slide readily over the skin. In getting the graft into the orbit, a small ball of gauze covered with crossed fiber rubber tissue was fashioned, and the graft was fitted on it before being pushed into the cavity. The graft took well on all tissues—bare bone, periosteum, fat, muscle and denuded subcutaneous tissue. The woman was ready to work in four weeks, the man in five. The production of pigment is much greater relatively in a slowly growing melanotic tumor in the living body than it is in a rapidly growing tumor in a younger person, just as it is in vitro.

DISCUSSION

DR. VIRGINIA KNEELAND FRANTZ (by invitation) (demonstration with lantern slides): This slide is from the first case, and I regret very much that there is not a section of all the tissue that Dr. Kirby removed. I did not realize at that time that we could get sections including the lids and the orbital tissues with adequate fixation for the globe at the same time, so I dissected most of the lid from the

globe in order to get satisfactory fixation, and the specimen lost some of its interest on that account.

This slide shows the actual section prepared for microscopic study. The tumor, as we found it, involved both the lids, especially at the inner canthi, and had grown around the conjunctival fold into the epibulbar tissues. The tumor is seen in the epibulbar tissue and comes quite close to the limbus. This area shows the inner surface of the lid, with the conjunctival epithelium, and the tumor is seen in the lid and in the margin. The eye has an open filtration angle and shows no evidence of glaucoma.

This is a photomicrograph to show the tumor in the lid. The conjunctival epithelium is intact, but the tumor has invaded the lid. It started on the outside of the lower lid as an epithelioma of the skin, but it has grown practically all the way through to the conjunctival surface on the inner side.

This section is to show the extension of the tumor from the epibulbar tissues down toward the limbus. It has grown as a rather superficial growth, as one would expect in a basal cell tumor; it has progressed toward the limbus, but it has not penetrated the globe.

This section shows the general topography of the tumor. It has grown more like a basal cell tumor with masses of cells surrounded on the outside by rather regularly arranged cylindric cells. In this section, in the center of the mass, there is a tendency toward the squamous type of cell, and in the next section there is a definite "pearl" with keratohyaline granules in the cells, so we must regard it as a mixed type of tumor, squamobasal. These tumors rather resemble the pure basal cell tumors in their long duration and slow growth and in the fact that they do not metastasize as pure squamous ones do.

In the next case the specimen was sectioned slightly more successfully, and the section shows the lids, the globe and the retrobulbar extension of the tumor. In this section the detachment of the lens is an artefact, but the detachment of the retina is what was observed clinically, and it was separated from the choroid by fluid. The tumor extends from just behind the equator of the globe around as a flat tumor, which undoubtedly was the reason for its late diagnosis; it extends into the ciliary body, abuts on the anterior chamber, and then grows as a flat growth along the anterior surface of the iris, and here is forming a pupillary membrane, a single strand of tumor cells. There are only six or seven cells in the section, and there are about three cells on the opposite side of the iris that morphologically resemble tumor cells, so that although it is difficult to be sure that we know a tumor cell at such distance from the rest of its fellows, we do feel that the tumor grew across, forming a pupillary membrane, and is encroaching on the other side of the iris.

This section does not show the optic nerve, and we have no section to show the extension of the tumor completely. In a series of sections it was obvious that the tumor had grown into the epibulbar tissues via the emissaria. It had surrounded the optic nerve, but had not invaded it, and it had formed this large mass, which was responsible for the man's marked exophthalmos. The tumor had extended into

the fat. It appeared encapsulated, but on microscopic section there were tumor cells free in the fat.

Dr. Kirby spoke about the large amount of melanin in these slowly growing tumors. When we sectioned the tumor, the melanin ran out like ink onto the section board.

These are the tumor cells along the anterior surface of the iris and in the anterior chamber, and here is the pupillary membrane, unfortunately not in high enough magnitude to confirm my word.

This is a slightly higher power view of the growth of the tumor on that side between the sclera and the ciliary body and encroaching on the iris, and this is the high power view that is characteristic of these tumors; they grow around the small blood vessels in a rather radial arrangement. They are composed of large spindle-shaped cells, with large vesicular nuclei and no mitoses to speak of, and the bizarre appearance is due to the melanin pigment in the cytoplasm of the tumor cells.

DR. ISADORE GOLDSTEIN: Ten years ago I did the same operation that Dr. Kirby described on a man who had a sarcoma of the globe involving the orbit. This method has two advantages: first, the short stay in the hospital, and second, if there is a new formation of the growth, you can readily recognize it through the smooth epithelial tissue.

DR. BERNARD SAMUELS: I remember Dr. Goldstein's case very well. The specimen was sent to the laboratory at the infirmary. I think that it would be a good idea for Dr. Goldstein to give a more accurate description of his operative technic, as he was the first one at the infirmary, and so far as I know the only one, to put in a graft immediately after the exenteration.

DR. ISADORE GOLDSTEIN: There was nothing unusual about the operation; the graft was taken from the thigh and laid on rubber tissue that was cut in four triangles, one placed above, one below, one temporally and one nasally, so that they overlapped each other. Then a piece of gauze was laid across the orbit, into which gauze was packed tightly, making a so-called Mikulicz packing. In another case, we did not use gauze, but used a waterbag. These grafts usually take. The apex is the only part that occasionally does not take; however, one can graft immediately should failure occur.

DR. DANIEL KIRBY: From the microscopic sections in the case of squamous cell epithelioma, one might wonder why it was necessary to remove the globe and to exenterate the orbit. The case was really worse than the microscopic sections make it appear. We saw a patient who had received a great deal of radium to destroy a growth, and it had almost entirely destroyed both eyes. It was necessary to remove one of the eyes because of the glaucoma and epitheliomatous invasion. I saw this patient this afternoon, and she still has actively growing squamous cell epithelioma about the eye and brow, and the result after irradiation has not been very good, so I thought that as we had a chance to remove the entire growth in this second patient, it was better to sacrifice the eye.

OPHTHALMOPLEGIA TOTALIS. DR. JOSEPH S. HORY.

M. F., a woman, aged 35, was first seen on May 18, 1931. For one month she had had severe neuralgia over the right side of the head. Four days before examination, ptosis of the right upper lid had developed. Examination showed complete ptosis of the right upper lid; the pupil was widely dilated, and no reactions were elicited. The eye was fixed. Vision in each eye was 20/20, the left eye showing no abnormality of structure. There were paralysis of the right palate and the vocal cords and a deviation of the tongue down to the right.

CYSTS OF THE CORNEA. DR. MILTON L. BERLINER.

Cysts of the cornea are very rare. In all, there have been about fifteen cases reported in the literature. I have been able to assemble fifteen additional cases from the extensive collections of microscopic preparations of the late Prof. Dr. Ernst Fuchs and Dr. Bernard Samuels. Only true corneal implantation cysts will be considered; that is, the cysts that have a definite lining membrane. The congenital cysts and the so-called dilated corneal lymph spaces will not be considered. These cysts may be classified according to location. There are three main types: (1) the precorneal, (2) the intracorneal and (3) the postcorneal. The precorneal cysts are those situated on the surface of the cornea or just below the epithelium, i. e., between the epithelium and Bowman's membrane. These cysts are generally formed by invaginations or implantations of the surface epithelium and occur in cicatrices after injuries, operations, e. g., removal of staphylomas, or following ulcers, particularly when the conjunctiva has become drawn over and is adherent to the cornea.

The intracorneal cysts are situated in the middle layers of the cornea between Bowman's membrane anteriorly and Descemet's membrane posteriorly. When situated in the cornea proper, they are very small, but if they extend into the sclera (corneoscleral type) they enlarge to a great size. These corneoscleral cysts often enlarge and extend posteriorly, encircling the globe by filling Tenon's space. Then they are known as peribulbar cysts. The postcorneal cysts are situated in the deep parts of the cornea and have to do with Descemet's membrane and the endothelium. Only one case has been reported, and I was able to find another. In these cases there is generally a splitting or a rupture of Descemet's membrane. This allows the endothelium to grow around the torn edges and line the corneal side of the detached Descemet's membrane, and also to continue over the posterior wall of the cornea. The gap is filled in by the formation of a new elastic membrane and a cyst results.

In addition, the cysts may be classified according to their lining. The precorneal cysts are generally lined by either corneal surface epithelium or by bulbar (conjunctival) epithelium. The intracorneal cysts are lined by either surface epithelium (corneal or bulbar) or by pigmented iris epithelium. The postcorneal cysts are always lined by endothelium.

Except for the large corneoscleral variety, these cysts are rarely diagnosed during life.

DISCUSSION

DR. BERNARD SAMUELS: I cannot add anything to what Dr. Berliner has said. He has given a splendid demonstration, and the classification is excellent.

What occurred to me was that all the cases that have been recorded are old; there has not been a single one reported in recent years. The sections from Vienna are from fifteen to twenty years old, and so are those from the infirmary. The reason that these cysts, which were always rare, are becoming rarer, I presume, is because we are handling injuries better than in former times, and another reason is that we are not excising staphylomas of the cornea, as we did in times past. Several of the cysts that Dr. Berliner showed developed after operations for staphylomas. In general, we are operating better than we used to.

SPONGIONEUROBLASTOMA OF THE OPTIC NERVE IN VON RECKLINGHAUSEN'S DISEASE. DR. ISADORE GOLDSTEIN.

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. DAVID WEXLER: The case that Dr. Goldstein presented has several points of interest. It is the first case in which a tumor resembling a glioma has been demonstrated pathologically which has not produced symptoms. As far as I can make out from the literature, the cases that have proved to be glioma or any other type of intradural nerve tumor have been suspected at first clinically, and no case has been discovered accidentally. The usual symptoms of exophthalmos, defects in vision and changes in the fundus were absent in this case. It must be a much more slowly growing tumor. It does not extend beyond the pia of the nerve, and does not invade the orbit to produce symptoms of pressure. I believe that it is also the first time a tumor of the optic nerve has been successfully demonstrated in association with a generalized neurofibromatosis.

Dr. Verhoeff, in his paper on intraneural nerve tumors published about ten years ago, admitted the possibility of gliomatous involvement of the optic nerve in association with peripheral neurofibromas, but he stated that positive evidence was still lacking. Several reported instances have failed of substantiation. The present case leaves no doubt of a new growth in the optic nerve in a case of generalized neurofibromatosis.

HEMORRHAGE WITHIN THE OPTIC NERVE SHEATH IN AN IRRADIATED FETUS. DR. DAVID WEXLER.

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. ARNOLD KNAPP: What was the period of time between the irradiation and the obtaining of the specimen?

DR. ISADORE GOLDSTEIN: The reason irradiation attacks the eye is because the eye is the first fully formed organ, and that is why the

other organs are not involved so early. Is it possible that the remaining ova are affected by the irradiation? If they are, and the women have children, what effect is it going to have on the eyes? Should opportunity present itself for such a study, a report will be made.

DR. DAVID WEXLER: The x-rays are applied at a single dose to the anterior and posterior part of the abdomen, some time during the second month of pregnancy. Spontaneous abortion usually occurs at from three and a half to four months, or about six weeks after irradiation. In two cases the fetuses have managed to survive until six and a half or seven months. This is quite rare, occurring probably once in 100 cases in which irradiation is applied. The eyes are fixed on an average of from one to two hours after the embryo is expelled from the uterus.

THE SYPHILITIC OPTIC ATROPHIES. DR. JOSEPH EARLE MOORE.

The rôle of syphilis in the etiology of the optic neuropathies was discussed, and the necessity was emphasized for careful and complete study of each patient as a diagnostic problem. The most important of the syphilitic optic atrophies is the so-called primary atrophy. Its relationship to tabes and to other forms of neurosyphilis was mentioned, and it was estimated that at any one time there are about 50,000 patients in the United States with this condition. Great emphasis was laid on the necessity for early diagnosis by means of cooperation between ophthalmologist and syphilologist, the routine ophthalmologic examination of all neurosyphilitic patients and the use of such diagnostic aids as carefully taken visual fields and tests of dark adaptation.

The pathology of the lesion was discussed, and it was pointed out that the process begins in the marginal fibers of the intracranial portion of the optic nerves anterior to the chiasm, but that there is as yet no agreement as to the relationship of degenerative to inflammatory processes, or as to the presence or absence of treponemas in the optic nerves.

The treatment for syphilitic primary optic atrophy prior to the introduction of the arsphenamines was hopeless, and even these drugs, when employed as a routine measure, did not materially improve the prognosis. Two modern methods of treatment, namely, subdural therapy and fever therapy, have been found in various parts of the world to be of some value in arresting the progress of the disease and in preserving useful vision. Lantern slides were shown, illustrating the application of these methods of treatment in individual patients, who were followed up for periods of from three to fourteen years. It was concluded that in a patient with early primary optic atrophy (vision in the better eye 20/40 or more), subdural treatment by various routes (intraspinal or intracisternal) and with various substances (arsphenaminized serum, neoarsphenamine, mercury or air), combined with appropriate general antisyphilitic treatment, offered at least a 50 per cent chance of arrest or even a slight improvement, and that by this means useful vision might be preserved for periods of time long exceeding the average expectancy of two years from the onset of symptoms. Fever therapy (malaria), while also successful in a somewhat smaller proportion of

cases, is more likely suddenly to extinguish the patient's remaining vision; it is more dangerous to life, and patients treated with it have not yet been followed for long enough periods to ensure permanency of the results. The choice of a method of treatment depends on the general physical and neurologic status of the patient, as well as on the condition of the eyes.

DISCUSSION

Dr. Mark Schoenberg: I am very happy to have heard this paper, which was so interesting and instructive. One of the reasons why we learn a great deal from such a paper is that Dr. Moore is not only a syphilographer, but he knows the medical and neurologic side of the question as well. He made a remark at dinner that he did not mention here, with which I thoroughly agree, and this remark is that so many men are misled by a 4+ Wassermann reaction; whenever they meet a neurologic or an ophthalmologic problem and the Wassermann reaction of the blood is 4+, they take it for granted that the problem is "syphilis," and they treat the condition as syphilis. The difficulty begins at the beginning, namely, how to diagnose a nonsyphilitic condition in a syphilitic patient.

I should like to ask Dr. Moore how he makes a diagnosis of a nonsyphilitic optic atrophy in the presence of a 4+ Wassermann reaction. I recall the classic case reported by Dr. Knapp several years ago, in a patient who had a 4+ Wassermann reaction and a number of other symptoms of syphilis, with failing vision and optic atrophy. This patient died, and at postmortem examination there were found a tumor of the meninges and a descending optic atrophy that looked like a primary optic atrophy. Some years ago one of my patients with a 4+ Wassermann reaction and a history of a primary lesion and secondary manifestations, whose impaired vision did not improve after a prolonged course of antisyphilitic treatment, responded promptly after he ceased smoking and drinking liquor. It was a case of toxic optic atrophy, a retrobulbar neuritis, in a patient with syphilis.

Probably it has been the experience of some of my colleagues, as it has been mine, that in the last few years the number of cases of primary optic atrophy has decreased; we also rarely see cases of interstitial keratitis. I should like to hear what the experience of Dr. Moore and of others has been regarding this disease. Does Dr. Moore prefer the use of bismuth to mercury, and has he had the feeling that mercury acts injuriously in some cases of primary optic atrophy? What is his routine technic in using arsphenamine and whatever else he uses —bismuth or mercury?

He reported a case of primary optic atrophy with very marked chorioretinitic changes. From the literature, as well as from my own experience, it seems that such occurrences are very rare.

I should also like to ask Dr. Moore whether he has had any experience with intracranial injections of corrosive mercuric chloride in cases of rapidly progressive optic atrophy and tabes.

Dr. Arnold Knapp: I wish to ask Dr. Moore two questions: 1. Does he base the indications for treatment on the inflammatory changes present in the spinal fluid? In other words, would he undertake

treatment intraspinally in a patient in whom the findings are practically negative as far as inflammatory changes are concerned? 2. Am I correct in my impression that he is not in favor of the malarial treatment?

DR. MARTIN COHEN: From the experience I have gathered in watching cases of optic atrophy, the pallor appears to me to be due frequently to a specific endarteritis involving the central artery. It appears to me that the color of the disk is not due to the proliferation of the tissue, but to a change in the vasculature that causes a certain amount of constriction of the small capillaries and brings about a distinct pallor of the disk, and that is the reason why Dr. Moore frequently cures a beginning case of apparent optic atrophy. I think that it is a degeneration of the axis cylinders, which condition results so frequently in total blindness. In optic atrophy there is a certain amount of improvement due to one drug or another, but it is a permanent condition, owing to the nature of the disease.

I believe that the point Dr. Schoenberg brought out concerning a decrease in the number of cases of interstitial keratitis and optic atrophy depends on how many patients one sees, how many clinics he is associated with, and how many clinics he actually attends. If you go to Central Islip or to Ward's Island, you will see a number of cases, indicating an increase in optic atrophy. Dr. Moore has said that from that standpoint syphilis is not increasing, but in such institutions I think that it is on the increase. As far as syphilis is concerned, these patients have been put on malarial treatment, and the results along those lines far exceed those from any other type of treatment.

DR. JOSEPH SHELDON SOMBERG: Continuing along the same lines that Dr. Cohen instituted, I might state that at the Manhattan State Hospital, where I am in charge of the eye service, we have around 8,000 psychiatric patients, about one half of whom have neurosyphilis and psychic symptoms, and therefore fall into the group with paresis. During the last nine years, we have seen about 300 cases of bilateral optic atrophy. We have come to the definite conclusion that subdural treatment is of no value and gives no results. We find that the best results are obtained with malarial treatment, which seems to work like clockwork for the mental disturbances as well. We have entirely discontinued the use of subdural treatment, for we believe that it is of value only when there is a meningeal type of syphilis, and we do not believe that the medication could reach the parenchymal type. The intravenous method is more apt to reach this type of syphilis, and we believe that a large proportion of the optic atrophies belong to the parenchymatous type of syphilis.

We also find that the atrophy of the optic nerve that we see at the Manhattan State Hospital, which of course is of the paretic type, does not usually progress to complete ophthalmologic blindness, that is, complete loss of light perception. There always seems to be some retention of vision, and various limits are seen in various cases. In some cases vision decreases to 20/40, in some to 20/50, in some to 20/70 and in others to 10/200, and sometimes it is even less. In the tabetic, but not in the paretic, type of atrophy, complete blindness is encountered. The

vision obtained remains stationary for years, and sometimes even shows a slight improvement, but progression is not very rapid in the paretic type. As stated, we now use malarial treatment and obtain very good results, and, as a rule, we find that the course of the optic atrophy is arrested almost instantaneously.

There is only one question that I wish to ask Dr. Moore, which is along the same line that Dr. Cohen brought up. What is the cause of the pallor of the optic nerve in which there are no functional disturbances?

Several years ago I presented a number of cases in which vision was normal and there was a marked pallor of the optic nerves. Is the pallor due to a disturbance of the vessels, or to some other cause? Dr. Cohen stated that in his opinion it is probably due to endarteritis of the central vessels. This is an interesting question, and I wonder whether Dr. Moore can answer it.

DR. JAMES W. SMITH: To what do you attribute the visual decrease in the patients treated with malarial therapy?

DR. JOSEPH EARLE MOORE: Dr. Schoenberg raised the question of the diagnosis of nonsyphilitic optic atrophy in a syphilitic patient. That, of course, is a matter of extraordinary difficulty. One occasionally finds a tumor of the brain in a syphilitic patient. We did make such a diagnosis last week, in a patient in whom we were fortunate enough to see complete destruction of the sella turcica by the x-rays. If it had not been for the x-rays, we would still be laboring under the impression that the patient had syphilitic optic atrophy. The only way one can approach the problem is by careful history taking and complete physical examination with the use of all accessory methods of diagnosis. Even then one will occasionally be completely mistaken and regard the patient as having a syphilitic optic atrophy merely because he has syphilis. I do not believe, however, that it is a frequent occurrence.

As to a possible decrease in the frequency of optic atrophy, which Dr. Schoenberg mentioned, I feel that there is no reliable evidence of such a decrease. Until recent months we have had no beds available for the treatment of patients with optic atrophy at Johns Hopkins Hospital. Since the opening of the new Osler Clinic, however, our own department has a number of beds for experimental purposes, which we are now devoting to the problem of the treatment for optic atrophy. When we first started, we had some difficulty in getting patients to fill the beds. At the same time, we have begun a routine ophthalmologic survey of all neurosyphilitic patients in the interests of early diagnosis, and Dr. Woods has been picking out a large number of patients whom he regards as having optic atrophy, often only on the basis of changes in dark adaptation or in the visual fields without much loss of visual acuity.

I have seen nothing to indicate that mercury has any particular toxic action on the optic nerve. Aside from atoxyl and the group of pentavalent arsenicals related to that drug, it seems to me that there is nothing to indicate that any drug that is used in the treatment for syphilis has a deleterious effect on the optic nerve or in the treatment for optic atrophy. We do prefer bismuth to mercury.

As to the question of the association of primary optic atrophy with pigmentary chorioretinitis, I realize that that is a very rare condition. It has been reported in three cases by Friedenwald in a recent number of the ARCHIVES. In this particular case, the diagnosis of pigmentary degeneration of the retina had been made.

I have had no personal experience with intraventricular injections. We are now treating patients intracisternally, with the idea that the substance injected may be placed in more close contact with the diseased tissue. There is a slight risk in doing cisternal punctures, whereas with spinal punctures there is no danger.

In answer to Dr. Knapp, we do not base the methods of treatment entirely on the evidence provided by the spinal fluid. A high cell count indicating an extreme degree of meningeal inflammation does not mean much more to us in the choice of a method of treatment than a very low cell count. We have in our series of patients a considerable number with negative spinal fluids, and we are now treating such patients more or less as an experiment.

The comments of Dr. Cohen and Dr. Somberg illustrate the extreme confusion that exists in the minds of all of us as to the pathology and diagnosis of optic atrophy. A French ophthalmologist, Abadie, has a theory that all primary optic atrophies are due to vasomotor disturbances of the central artery of the nerve, and he treats his patients with belladonna. He maintains that he gets beneficial results, but he has published no substantiating details as to whether one can detect color changes in the optic nerve due to constriction of the vessels. The diagnosis of optic atrophy in our own material has been made, not by ourselves, but by the ophthalmologists of the Wilmer Institute. It seems to me that Dr. Somberg's material must be somewhat unusual if he has never seen a patient with syphilitic optic atrophy go blind, because that is the usual course of events in Baltimore. As to the patients Dr. Somberg treated some years ago, and on whom he reported apparently contradictory findings between the degree of pallor of the optic nerve and the visual acuity of the patient, I gathered from reading his article that most of these patients were under treatment with tryparsamide. This drug does have deleterious effects on the optic nerve. Dr. Woods and I reported the cases of various patients in whom such difficulties arose, and it has occurred to me to wonder whether that drug might not be responsible for the loss of visual acuity which some of Dr. Somberg's patients subsequently showed.

As to the treatment of patients with early optic atrophy, we shall be quite willing to meet the criticism that the patients did not have primary optic atrophy, provided we can prevent loss of vision. By this I mean that I would far rather have a patient who retains useful vision, even if the diagnosis is incorrect, than to have a patient who becomes completely blind and be right about it. The crux of the whole matter seems to lie in the question of improvement in the methods of early diagnosis. We must get the patients and recognize the condition before they stumble into the clinic with vision of 5/200 in each eye. If we do not get them sooner than that, little or nothing can be done. Perhaps this will involve education among the laity. Unfortunately, patients with failing vision often go first to an optometrist or optician,

and by the time they get to the ophthalmologist, vision is often so seriously impaired that little can be done about it. How that question can best be approached is a subject with which you are far more familiar than I am. I do feel that it is worth while to emphasize the necessity for study of the points that will lead us to better diagnosis and the application of one or another method of treatment that has been demonstrated to be of some value.

Our experience with malarial treatment is relatively limited. We have treated only about two dozen patients, and my comments about it are mainly based on papers from the literature, from which three facts appear: 1. In a patient with a progressive optic atrophy, malaria is much more likely to produce sudden visual failure during treatment than any other form of treatment. 2. It is a method of treatment more dangerous to life than any other method. 3. The good results reported from its use have been mostly in patients who have been followed up for only a short period after treatment. The literature is filled with papers reporting successful results in the cases of patients whose vision had not become worse during the six weeks of treatment. When reports begin to appear which talk in terms of six years, I shall be more convinced.

It is worth while emphasizing the fact that subdural treatment is a relatively innocuous method, so far as the patient's life is concerned, whereas malaria carries with it a distinct risk to life. In choosing any one method, one must take into consideration not only the state of the patient's eyes, but also his general physical and neurologic status.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

ELMORE BREWERTON, F.R.C.S., *President, in the Chair*

June 12, 1931

MACULAR DETACHMENT IN IRIDOCYCLITIS. MR. EUGENE WOLFF.

As long ago as 1901, Fuchs drew attention to the fact that edema of the macula might result from inflammatory lesions of the anterior portion of the eye. In cases of perforating injury with infection, it could be seen that the disease of the posterior segment of the eye commenced with an extensive immigration of leukocytes from the retinal vessels, so that the inner surface of the retina might be covered by a purulent exudate, while its outer surface and the choroid might be normal. This purulent retinitis led to panophthalmitis. So it seemed that the bacteria or their toxins had passed from the infected anterior portion of the eye through the vitreous and acted directly on the inner layers of the retina, producing in that way an intense leukocytosis. With a less intense toxin there might not be pus formation, or even a wandering out of leukocytes—nothing more than a transudation of serum. With a still less intense toxin there might only be inflammatory edema at the macula, which was exceptionally vulnerable.

Why was the macular region particularly vulnerable? Fuchs said that it was because of its delicate structure, while Iwanoff held that edema of the retina started in situations where the capillaries ended in circular anastomoses, i. e., on each side of the outer plexiform layer, at the ora serrata, in the neighborhood of the papilla and around the fovea centralis. Zeeman pointed out that the retinal venules at the ora serrata had at first a circular course and then bent radially toward the disk, and that these venules received tributaries from behind. The outer layers of the retina were supplied by the choriocapillaris, and the retinal vessels reached only the outer side of the inner nuclear layer, leaving the outer molecular layer between the two regions avascular, and so they were only slightly resistant to injury, and were very liable to degeneration. But it had also been suggested that the macula was most affected because the neuro-epithelium was almost next to the toxin-containing vitreous, and the avascularity of the region rendered the absorption of toxin difficult.

One aspect of the subject, however, seemed to have received but little attention. In a paper (by Dr. Francis Davis and himself) which Mr. Wolff recently read before the Ophthalmological Society he gave structural reasons for the special site of onset of papilledema, and for the distribution in the retina of the edematous fluid associated with papilledema. It was there suggested that the whole area drained by the central vein must contain an excess of fluid, i. e., edema. The amount in each portion must depend on the density of the structure of the region in question.

The nerve fibers at the disk had a relatively loose arrangement, and much fluid would collect and produce obvious swelling, whereas the lamina cribrosa, with its dense meshwork of fibrous trabeculae, contained but little excess fluid in papilledema. The author suggested also that one of the most important factors in the vulnerability of the macula and its liability to be detached was the structure of Henle's fiber layer and its well known tendency to swell.

The author suggested that in iridocyclitis and other inflammatory conditions of the anterior portion of the eye, the toxins or bacteria passed back into the vitreous, and came into contact with the whole retina. The macula would be especially affected, because the toxin acting here would produce a great accumulation of fluid in Henle's fiber layer. This fluid would remain unabsorbed longer than elsewhere, because of the vascular arrangements, which would be further embarrassed by the pressure of the fluid. The fluid would affect vision, not only by its presence in the region of most acute vision, but by pressure of the contained toxins on contiguous structures. At a later stage an albuminous exudate might form between the neuro-epithelium and the pigment layer, and produce a true detachment. In two of the cases where this occurred, a true detachment was present only in the macular region.

But the macula might be affected from the choroidal side, though in the majority of instances this did not appear to be the case. Zeeman considered that the inflammation passed from the pars plana of the ciliary body directly into the retina, reaching the posterior portion of the eye by traveling back along the retinal vessels.

In all the cases in which there was a swollen macula there was an infiltration of leukocytes at the nerve head; this, with the accompanying inflammatory exudate, was probably responsible for producing traumatic or perforative papilledema, which could sometimes be noted during life.

The cases were described in detail.

DISCUSSION

MR. E. TREACHER COLLINS: Unless the fixation were done very soon after excision, rucking at the back of the macula might occur, as it was a very early and prominent postmortem change. It was quite possible that such appearances might be artefacts.

MR. E. WOLFF: It is difficult to make a normal section of the outer fiber layer and of Henle's fiber layer. The specimens in all the cases were fixed in Zengler's acetic acid solution immediately after removal of the eye.

MR. GAYER MORGAN: The question was asked whether Mr. Wolff had investigated hypopyon ulcer in this way. In many cases after hypopyon ulcer one got a good view of the macula, and vision was very poor; if any case would answer the description of the passage of toxins from front to back, these would.

MR. E. WOLFF: The cases mentioned by Mr. Morgan were considered excellent for the purpose, but I have not looked for edema of the macula in eyes with hypopyon ulcer which have been excised.

Book Reviews

FOURTH ANNUAL REPORT OF THE GIZA MEMORIAL OPHTHALMIC LABORATORY, CAIRO. Pp. 139. Cairo, Egypt: Schindler's Press, 1930.

This splendid institution has continued its valuable work, and this report, like its predecessors, will repay careful study. Interesting and unusual cases encountered during the year are reported in the sections on pathology and clinical ophthalmology, but most attention is devoted to the problem of trachoma. In former years investigation was greatly hampered by the lack of follow-up facilities, as the fellahin seldom cooperated in this respect. The data obtained were therefore incomplete, and the conclusions drawn were necessarily inaccurate. It was then decided to select a village so situated that it represented a typical cross-section of Egypt as a whole and to install a resident ophthalmologist. The resulting report is a splendid effort. It does not lend itself to condensation, but is worth the attention of those who are interested in trachoma and the purulent ophthalmias.

Investigation in the laboratory itself was rewarded by several interesting observations. The presence of normal follicles in the conjunctiva of monkeys, which was suggested in last year's report, was definitely confirmed. This folliculosis varies from time to time without treatment and may be induced by mere irritation and trauma, even under aseptic conditions. It will be seen how difficult it is to interpret observations when one is working on the conjunctiva of monkeys. Experimental inoculations with three strains of *B. granulosis* (Noguchi), which were sent from the Rockefeller Foundation in New York, gave entirely negative results, even when the organisms were inoculated into the blind human eye, as was done in one case. *B. granulosis* has not yet been isolated from Egyptian cases of trachoma.

Investigation into the serologic reactions in trachoma did not produce conclusive results.

Treatment for trachoma by intravenous injections of copper ammonium sulphate is reported as unsatisfactory, and the attempts to clear corneal nebulae by subconjunctival injections of phenolaine have been abandoned.

G. M. BRUCE.

NATIONAL MEDICAL JOURNAL OF CHINA. Volume 17. Pp. 166. Peiping, China: Peiping University Medical College, February, 1931.

This is a memorial number in honor of Professor Fuchs. It begins with an obituary by Sallmann. The results of several investigations are reported, including the effects of insufflations of air into the anterior chamber and the relative value of oily and aqueous solutions of miotic and mydriatic drugs. Another study reveals the efficacy of radium in closing recent corneal fistulas. There are articles on western ophthalmology in China and on ancient Chinese ophthalmology. Reports of cases include such diseases as xanthomatosis bulbi, hydrophthalmos in relation to facial nevi, chancre of the retrotarsal folds, ocular neoplasms, the formation of blood vessels in the vitreous and injury of the cornea by native gunpowder. The usual annotations and book reviews bring this excellent volume to a close. It is a worthy tribute to the man in whose memory it is published.

G. M. BRUCE.

Directory of Ophthalmologic Societies *

FOREIGN

British Medical Association, Section on Ophthalmology

President: Mr. E. E. Maddox, Glenartney, Poole Road, Bournemouth, England.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 7-9, 1932.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 Avenue de La Motte Picquet, Paris 7è.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: New Orleans. Time: 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. F. Barnhill, Miami Beach, Fla.
President-Elect: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: French Lick, Indiana. Time: Sept. 14-19, 1931.

American Ophthalmological Society

President: Dr. Edward C. Ellett, 130 Madison Ave., Memphis, Tenn.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.
Place: New London, Conn. Time: June, 1932.

SECTIONAL

New England Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Me.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Chester H. Bowers, 1136 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Sioux Valley Eye and Ear Academy

President: Dr. Claude T. Uren, City National Bank Bldg., Omaha.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Fontanelle Hotel, Omaha. Time: November, 1931.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

Southwestern Michigan Triological Society

President: Dr. C. Ward Ellis, 215 N. Walnut St., Lansing.
Secretary: Dr. H. B. Weinburgh, 301 Seymour St., Lansing.
Time: Third Thursday of alternate months.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: First floor, Metropolitan Building, Denver. Time: 7:30 p. m., third Saturday of the Month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. Gordon F. Harkness, 215 Main St., Davenport.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Douglas F. Wood, 74 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.

Montana Academy of Oto-Ophthalmology

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: W. L. Diven, National Bank Bldg., Bismarck.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. M. Hendershott, 193, 11th St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

Utah Ophthalmological Society

President: Dr. Franklin H. Raley, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. H. Leroy Smith, 1005 Medical Arts Bldg., Salt Lake City.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. C. P. Jones, Newport News.
Secretary-Treasurer: Dr. Henry Grant Preston, 307 Professional Bldg., Harrisonburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. J. Wallace Hurff, 86 Washington St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

Atlanta Academy of Ophthalmology and Oto-Laryngology

President: Dr. John R. Childs, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society. Section on Ophthalmology

Chairman: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. A. F. Luhr, 40 W. North Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. E. Burner, Guardian Bldg., Cleveland.
Secretary: Dr. M. P. Motto, Rose Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadephia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. A. M. Hauer, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. M. Sellers, 717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831, 5th Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. J. J. Richardson, 606 Penn St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, 1304 Medical Arts Bldg., Forth Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robison, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. R. R. Montgomery, 102 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Russell T. Uhls, 823 Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Oto-Laryngology

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Herbert F. Wolters, 130 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. William P. Haney, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. A. Cassiday, 1620 Medical Arts Bldg., Omaha.
Place: Medical Tea Room. Time: 5:15 and 7:00 p. m., third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Emanuel U. Wallerstein, Professional Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Martin H. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott C. Applewhite, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. W. F. Swett, 490 Post St., San Francisco.
Secretary: Dr. Robert C. Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.